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 NEWS  1          Web Page for STN Seminar Schedule - N. America
 NEWS  2  MAY 01  New CAS web site launched
 NEWS  3  MAY 08  CA/CAplus Indian patent publication number format defined
 NEWS  4  MAY 14  RDISCLOSURE on STN Easy enhanced with new search and display
                  fields
 NEWS  5  MAY 21  BIOSIS reloaded and enhanced with archival data
 NEWS  6  MAY 21  TOXCENTER enhanced with BIOSIS reload
 NEWS  7  MAY 21  CA/CAplus enhanced with additional kind codes for German
                  patents
 NEWS  8  MAY 22  CA/CAplus enhanced with IPC reclassification in Japanese
                  patents
 NEWS  9  JUN 27  CA/CAplus enhanced with pre-1967 CAS Registry Numbers
 NEWS 10  JUN 29  STN Viewer now available
 NEWS 11  JUN 29  STN Express, Version 8.2, now available
 NEWS 12  JUL 02  LEMBASE coverage updated
 NEWS 13  JUL 02  LMEDLINE coverage updated
 NEWS 14  JUL 02  SCISEARCH enhanced with complete author names
 NEWS 15  JUL 02  CHEMCATS accession numbers revised
 NEWS 16  JUL 02  CA/CAplus enhanced with utility model patents from China
 NEWS 17  JUL 16  CAplus enhanced with French and German abstracts
 NEWS 18  JUL 18  CA/CAplus patent coverage enhanced
 NEWS 19  JUL 26  USPATFULL/USPAT2 enhanced with IPC reclassification
 NEWS 20  JUL 30  USGENE now available on STN
 NEWS 21  AUG 06  CAS REGISTRY enhanced with new experimental property tags
 NEWS 22  AUG 06  BEILSTEIN updated with new compounds
 NEWS 23  AUG 06  FSTA enhanced with new thesaurus edition
 NEWS 24  AUG 13  CA/CAplus enhanced with additional kind codes for granted
                  patents

 NEWS EXPRESS  29 JUNE 2007:  CURRENT WINDOWS VERSION IS V8.2,
               CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS LOGIN    Welcome Banner and News Items
 NEWS IPC8     For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that
specific topic.

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FILE 'HOME' ENTERED AT 15:08:54 ON 13 AUG 2007

=> file caplus, stn, biosis
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 STNGUIDE      - Descriptive information about STN databases
 STNMAIL       - STN Electronic Mail Service
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accessing the remaining files of your multiple file entry.
ENTER A FILE NAME OR (IGNORE):emd
'EMD' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):end

=> file caplus medline biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'CAPLUS' ENTERED AT 15:09:36 ON 13 AUG 2007
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FILE 'MEDLINE' ENTERED AT 15:09:36 ON 13 AUG 2007

FILE 'BIOSIS' ENTERED AT 15:09:36 ON 13 AUG 2007
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=> s (glucosamin? or mannosamin?) and (bambuterol or bitolterol or carbuterol or
clenbuterol or clorprenaline or dioxethedrine)
L1            12 (GLUCOSAMIN? OR MANNOSAMIN?) AND (BAMBUTEROL OR BITOLTEROL OR
                 CARBUTEROL OR CLENBUTEROL OR CLORPRENALINE OR DIOXETHEDRINE)

=> s (glucosamin? or mannosamin?) and (dopexamine or ephedrine or epinephrine or
etafedrine or ethylnorepinephrine or "ethyl norepinephrine" or fenoterol or
formoterol or hexoprenaline))
UNMATCHED RIGHT PARENTHESIS 'PRENALINE))'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (glucosamin? or mannosamin?) and (dopexamine or ephedrine or epinephrine or
etafedrine or ethylnorepinephrine or "ethyl norepinephrine" or fenoterol or
formoterol or hexoprenaline)
L2           129 (GLUCOSAMIN? OR MANNOSAMIN?) AND (DOPEXAMINE OR EPHEDRINE OR
                 EPINEPHRINE OR ETAFEDRINE OR ETHYLNOREPINEPHRINE OR "ETHYL NOREP
                 INEPHRINE" OR FENOTEROL OR FORMOTEROL OR HEXOPRENALINE)

=> s (glucosamin? or mannosamin?) and (isoetarine or isopreterenol or mabuterol or
malbuterol or metaproterenol or methoxyphenamine or pirbuterol or procaterol or
protokylol or reproterol or rimiterol)
L3            6 (GLUCOSAMIN? OR MANNOSAMIN?) AND (ISOETARINE OR ISOPRETERENOL
                 OR MABUTEROL OR MALBUTEROL OR METAPROTERENOL OR METHOXYPHENAMINE
                 OR PIRBUTEROL OR PROCATEROL OR PROTOKYLOL OR REPROTEROL OR RIMIT
                 EROL)

=> s (glucosamin? or mannosamin?) and (ritodrine or salbutamol or salmeterol or
soterenol or terbutaline or tretoquinol or tulobuterol)
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L4            9 (GLUCOSAMIN? OR MANNOSAMIN?) AND (RITODRINE OR SALBUTAMOL OR
               SALMETEROL OR SOTERENOL OR TERBUTALINE OR TRETOQUINOL OR TULOBUT
              EROL)

=> dup rem l1-l4
L*** HAS NO ANSWERS
L*** HAS NO ANSWERS
'144' ANSWERS REMOVED DUE TO ANSWER OVERLAP
PROCESSING COMPLETED FOR L1
PROCESSING COMPLETED FOR L2
PROCESSING COMPLETED FOR L2
PROCESSING COMPLETED FOR L2
PROCESSING COMPLETED FOR L2
PROCESSING COMPLETED FOR L3
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PROCESSING COMPLETED FOR L3
PROCESSING COMPLETED FOR L4
PROCESSING COMPLETED FOR L4
PROCESSING COMPLETED FOR L4
PROCESSING COMPLETED FOR L4
L5           106 DUP REM L1-L4 (50 DUPLICATES REMOVED)

=> s l5 and py<=2003
   2 FILES SEARCHED...
L6            82 L5 AND PY<=2003

=> d l5 1-106 ibib abs

L5   ANSWER 1 OF 106  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2007:150999  CAPLUS
DOCUMENT NUMBER:          146:224347
TITLE:                    Fluorescent sensors with coumarin aldehyde or
                          quinolone aldehyde core for cellular amines detection
INVENTOR(S):              Glass, Timothy; Gillis, Kevin; Secor, Kristen
PATENT ASSIGNEE(S):       The Curators of the University of Missouri, USA
SOURCE:                   PCT Int. Appl., 54pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.            KIND   DATE        APPLICATION NO.          DATE
     ----------------      ----   --------    ---------------------    --------
     WO 2007016495         A2     20070208    WO 2006-US29760          20060728
     WO 2007016495         A3     20070607
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
             KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
             MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU,
             SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG,
             US, UZ, VC, VN, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
             CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
             GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRIORITY APPLN. INFO.:                       US 2005-704012P      P  20050729
                                             US 2005-735695P      P  20051110
OTHER SOURCE(S):          MARPAT 146:224347
```

AB The invention provides compds. of formulas of coumarin-, quinolone-, and quinolone-dimer-based sensors, methods of making them, and methods of their use. The compds. of the invention can be used as fluorescent sensors, for example, to detect an amine-containing analyte in a biol. sample. The compds. can be selective for one type of amine over others and the amount of fluorescence can be correlated with the concentration of the amine in the sample.

L5 ANSWER 2 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:817006 CAPLUS
TITLE: Stable therapeutic formulations
INVENTOR(S): Ameri, Mahmoud; Cormier, Michel J. N.; Sellers, Scott; Maa, Yuh-Fun
PATENT ASSIGNEE(S): Alza Corp., USA
SOURCE: PCT Int. Appl., 50pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007084247	A2	20070726	WO 2006-US49488	20061228
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2007184096	A1	20070809	US 2006-617639	20061228
PRIORITY APPLN. INFO.:			US 2005-754948P P	20051228

AB Compns. of and methods for formulating and delivering biol. active agent formulations having enhanced phys. stability, and wherein deterioration from the presence of oxygen and/or water is minimized and/or controlled, to yield a stable formulation are claimed. The compns. of and methods for formulating and delivering biol. active agents of the present invention further facilitate their incorporation into a biocompatible coating which can be employed to coat a stratum corneum piercing microprojection, or a plurality of stratum corneum piercing microprojections of a delivery device, for delivery of the biocompatible coating through the skin of a subject, thus providing an effective means of delivering the biol. active agents. A delivery device having stratum corneum piercing microprojections coated with a formulation of hPTH (1-34)was prepared The primary packaging for all dosages of the systems was a heat sealed foil pouch purged with nitrogen gas. The moisture and oxygen levels were substantially reduced in the packages.

L5 ANSWER 3 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:590568 CAPLUS
DOCUMENT NUMBER: 147:31134
TITLE: Novel use of thienopyrimidines and their preparation, pharmaceutical compositions and use in the treatment of inflammatory diseases
INVENTOR(S): Taylor, Steven; Coulter, Thomas Stephen; Jaekel,

Stefan; Aicher, Babette; Kelter, Arndt-Rene; Kraemer, Joachim; Kirchhoff, Christian; Scheel, Andreas; Woelke, Julian

PATENT ASSIGNEE(S): Developen Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 86pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

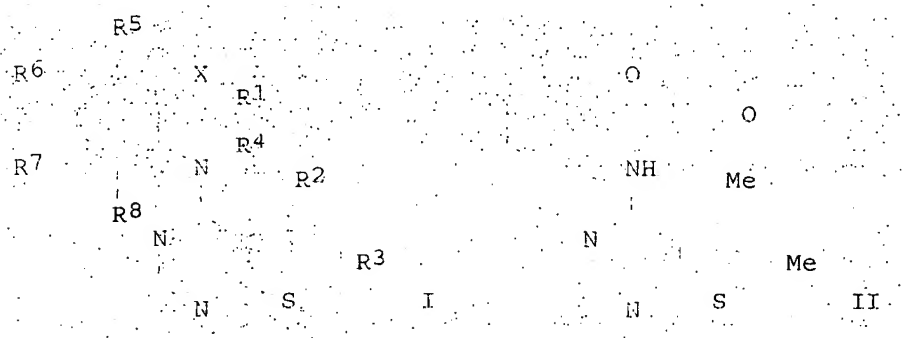
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007059905	A2	20070531	WO 2006-EP11081	20061117

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: EP 2005-25772 A 20051125
OTHER SOURCE(S): MARPAT 147:31134

GI



AB The invention relates to the use of thienopyrimidine compds. of formula I for the production of pharmaceutical compns. for the treatment of inflammatory diseases. Compds. of formula I wherein X is O, S, SO2, (un)substituted CH2; CH(halo) C(halo)2, CO, CONH2 and derivs., and NH and derivs.; R1 is H, C1-6 alkyl, C1-6 alkyl-C3-10 (hetero)cycloalkyl, 3- to 10-membered (hetero)cycloalkyl, C6-10 aryl, etc.; R2 and R3 are independently H, C1-6 alkyl, C1-6 alkyl-C3-10 cycloalkyl, C3-10 cycloalkyl, C6-10 aryl, etc.; R4 is H, C1-4 alkyl, urea, thiourea, and (un)substituted acetyl; R4X may form a 5- tp 6-membered heterocyclic ring; R5 and R7 are independently H, halo, CN, CO2H and derivs., OH and derivs., CONH2 and derivs.m SO2NH2 and derivs. SO2H and derivs., etc.; R6 and R8 are independently H, halo, CN, CO2H and derivs., OH and derivs., CONH2 and derivs.; SO2NH2 and derivs., SO2H and derivs., etc.; and their metabolites, prodrugs, pharmaceutically acceptable salts thereof, are claimed. Example compound II was prepared by etherification of 2-fluoronitrobenzene with 3-hydroxytetrahydrofuran; the resulting 3-(2-nitrophenoxy)tetrahydrofuran underwent hydrogenation to give 2-(tetrahydrofuran-3-yloxy)phenylamine, which underwent arylation

with 4-chloro-5,6-dimethylthieno[2,3-d]pyrimidine to give compound II. All the invention compds. were evaluated for their kinase inhibitory activity.

L5 ANSWER 4 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1354113 CAPLUS
DOCUMENT NUMBER: 146:94673
TITLE: Screening for specific cytochrome P 450 proteins or substrates and modification of cytochrome P 450 substrates using transgenic plants
INVENTOR(S): Kruse, Tanya; Park, Joon-Hyun; Bobzin, Steven Craig
PATENT ASSIGNEE(S): Ceres Inc., USA
SOURCE: PCT Int. Appl., 1432pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006138012	A1	20061228	WO 2006-US19001	20060517

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2005-691956P P 20050617
AB Methods and materials for modifying candidate compds. utilizing transgenic plant cells capable of expressing a cytochrome P 450 or related polypeptide are disclosed. Materials and methods of screening for a substrate of a P 450 and for screening a collection of polypeptides for a P 450 capable of modifying a compound are also disclosed. Screening for a substrate of a P 450 comprises contacting a pharmacol. active candidate compound with plant cells containing a recombinant nucleic acid construct expressing a P 450 polypeptide and determining whether the candidate compound is
modified after the contact. Similarly, a collection of P 450's can be screened for a P 450 capable of modifying a compound having a pharmacol. activity. The invention provides 137 Arabidopsis thaliana cytochrome P 450-encoding nucleic acids and their orthologs from other plant species.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:101295 CAPLUS
DOCUMENT NUMBER: 144:164267
TITLE: Physiologically active composition including davidigenin and other compounds, as well as glycosides thereof, and use in the treatment of diabetes and related conditions
INVENTOR(S): Jaeger, Ralf; Wenk, Hans-Henning; Dieck, Heike Tom; Hoppe, Hans Ullrich; Rabeler, Roland
PATENT ASSIGNEE(S): Bioghurt Biogarde Gmbh & Co. KG, Germany
SOURCE: PCT Int. Appl., 23 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006010560	A2	20060202	WO 2005-EP7964	20050721
WO 2006010560	A8	20060330		
WO 2006010560	A3	20060601		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SZ, BE, CY, FR, GR, IE, IT, MC, NL, SI, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
DE 102004036047	A1	20060223	DE 2004-102004036047	20040724
AU 2005266545	A1	20060202	AU 2005-266545	20050721
CA 2574894	A1	20060202	CA 2005-2574894	20050721
EP 1784201	A2	20070516	EP 2005-776027	20050721
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				

PRIORITY APPLN. INFO.: DE 2004-102004036047A 20040724
WO 2005-EP7964 W 20050721

AB The invention discloses a physiol. active composition containing effective quantities of at least one compound from the series 4-O-methyldavidigenin, 4'-O-methyldavidigenin, davidigenin, elemicin, isoelemicin, herniarin, demethoxycapillarisin, particularly 6-demethoxycapillarisin and/or 6-demethoxy-3'-methoxycapillarisin, hispiludin and 9-hydroxy-10E,12Z,15Z-octadecatrienoic acid, and their glycosides, particularly glucosides and/or rhamnoglucosides of 4-O-methyldavidigenin and/or 4'-O-methyldavidigenin, salts and derivs. The composition of the invention may be used for the treatment of diabetes and related conditions.

L5 ANSWER 6 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:269132 CAPLUS
DOCUMENT NUMBER: 144:338068
TITLE: Composition and method to optimize and customize nutritional supplement formulations by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes
INVENTOR(S): Blum, Kenneth; Meshkin, Brian; Downs, Bernard William
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 80 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006062859	A1	20060323	US 2005-197980	20050805

PRIORITY APPLN. INFO.: US 2004-599829P P 20040805

AB The invention relates to a composition and custom business model and methods to measure genetic and metabolomic contributing factors affecting disease diagnosis, stratification, and prognosis, as well as the metabolism, efficacy and/or toxicity associated with specific vitamins, minerals, herbal supplements, homeopathic ingredients, and other ingredients for the purposes of customizing a subject's nutritional supplements with custom formulations to optimize health outcomes. For example, Synaptose formula comprised DLPA 2000 mg, various glyconutrients including, but not limited to one or more of glucose, fucose, mannose, galactose, xylose, N-acetylglucosamine, N-acetylgalactosamine, and N-acetylneuraminic acid (sialic acid), all of which can be present as either monosaccharides or complexed as oligo- and/or polysaccharides in various foodstuffs including but not limited to aloe, fenugreek, numerous species of medicinal mushrooms, western larch (primarily tree sap and bark), and many more glycoside-rich botanical substances. In addition, the formula contained arabinose, arabinogalactans, and other polyglycan-rich substances, L-glutamine 150 mg, L-tyrosine 750 mg, 5-hydroxytryptophan 100 mg, chromium salt up to 1000 μg or more, Rhodiola rosea 200 mg, passion flower 100 mg, vitamin B6 20 mg, magnolia flower 20 mg, minerals (calcium 275-750 mg, magnesium at least 100-750 mg, and potassium at least 250-2000 mg), and salts of (-)-hydroxycitric acid up to 3000 mg.

L5 ANSWER 7 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1185811 CAPLUS
DOCUMENT NUMBER: 146:117217
TITLE: Chemical screening methods to identify ligands that promote protein stability, protein crystallization, and structure determination
AUTHOR(S): Vedadi, Masoud; Niesen, Frank H.; Allali-Hassani, Abdellah; Fedorov, Oleg Y.; Finerty, Patrick J., Jr.; Wasney, Gregory A.; Yeung, Ron; Arrowsmith, Cheryl; Ball, Linda J.; Berglund, Helena; Hui, Raymond; Marsden, Brian D.; Nordlund, Paer; Sundstrom, Michael; Weigelt, Johan; Edwards, Aled M.
CORPORATE SOURCE: Structural Genomics Consortium, Univ. Toronto, Toronto, ON, M5G 1L5, Can.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2006), 103(43), 15835-15840
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The 3D structures of human therapeutic targets are enabling for drug discovery. However, their purification and crystallization remain rate determining In individual cases, ligands have been used to increase the success rate of protein purification and crystallization, but the broad applicability of this approach is unknown. The authors implemented two screening platforms, based on either fluorometry or static light scattering, to measure the increase in protein thermal stability upon binding of a ligand without the need to monitor enzyme activity. In total, 221 different proteins from humans and human parasites were screened against one or both of two sorts of small-mol. libraries. The first library comprised different salts, pH conditions, and commonly found small mols. and was applicable to all proteins. The second comprised compds. specific for protein families of particular interest (e.g., protein kinases). In 20 cases, including nine unique human protein kinases, a small mol. was identified that stabilized the proteins and promoted structure determination The methods are cost-effective, can be implemented in any laboratory, promise to increase the success rates of purifying and crystallizing human proteins significantly, and identify new

ligands for these proteins.
REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:983611 CAPLUS
DOCUMENT NUMBER: 143:292527
TITLE: Bioavailability and improved delivery of alkaline pharmaceutical drugs
INVENTOR(S): Yu, Ruey J.; Van Scott, Eugene J.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 16 pp., Cont.-in-part of U.S. Ser. No. 792,273.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005196418	A1	20050908	US 2005-50434	20050204
US 2004214215	A1	20041028	US 2004-792273	20040304
WO 2006084174	A2	20060810	WO 2006-US3917	20060206

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.:		
US 2004-792273	A2	20040304
US 2003-452557P	P	20030307
US 2005-50434	A	20050204

OTHER SOURCE(S): MARPAT 143:292527
AB Embodiments of the invention relate to a composition, a process of making the composition, and to the use of the composition. The compns. include a mol. complex formed between an alkaline pharmaceutical drug and at least one selected from a hydroxy acid, a polyhydroxy acid, a related acid, a lactone, or combinations thereof. The compns. provide improved bioavailability and improved delivery of the drug into the cutaneous tissues. For example, diphenhydramine hydrochloride 29 g (0.1 mol) was dissolved in water and 5 N sodium hydroxide generating diphenhydramine free base. Gluconolactone 18 g (0.1 mol) was added to form a mol. complex of 0.1 mol diphenhydramine free base with 0.1 mol gluconic acid/gluconolactone. The solution thus obtained was used for various forms of topical formulations including oil-in-water creams, lotions, gels and solns.

L5 ANSWER 9 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:614580 CAPLUS
DOCUMENT NUMBER: 143:139175
TITLE: Frequency-assisted transdermal agent delivery method and system
INVENTOR(S): Chan, Keith T.; Cormier, Michel J. N.; Lin, WeiQi
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 24 pp.

CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005153873	A1	20050714	US 2004-971441	20041021
AU 2004314416	A1	20050804	AU 2004-314416	20041021
WO 2005069758	A2	20050804	WO 2004-US34923	20041021
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
BR 2004017757	A	20070410	BR 2004-17757	20041021
JP 2007519446	T	20070719	JP 2006-549239	20041021

PRIORITY APPLN. INFO.: US 2004-535275P P 20040109
WO 2004-US34923 W 20041021

AB The invention discloses an apparatus and method for transdermally delivering a biol. active agent comprising a delivery system having a microprojection member (or system) that includes a plurality of microprojections (or array thereof) that are adapted to pierce through the stratum corneum into the underlying epidermis layer, or epidermis and dermis layers, a formulation containing the biol. active agent and an oscillation-inducing device. In one embodiment, the biol. active agent is contained in a biocompatible coating that is applied to the microprojection member. In a further embodiment, the delivery system includes a gel pack having an agent-containing hydrogel formulation that is disposed on the microprojection member after application to the skin of a patient. In an alternative embodiment, the biol. active agent is contained in both the coating and the hydrogel formulation.

L5 ANSWER 10 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:588290 CAPLUS
DOCUMENT NUMBER: 143:103249
TITLE: Composition and method for healing tissues
INVENTOR(S): Petito, George D.; Petito, Anita M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 13 pp., Cont.-in-part of U.S. Ser. No. 457,599.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005147679	A1	20050707	US 2005-71464	20050304
US 6476005	B1	20021105	US 1999-360169	19990726
US 2002025921	A1	20020228	US 2001-983274	20011023
US 2003212005	A1	20031113	US 2003-457599	20030610

PRIORITY APPLN. INFO.: US 1998-46710 B2 19980324
US 1999-360169 A2 19990726

US 2001-983274 B1 20011023
US 2003-457599 A2 20030610

AB The composition and method for healing tissues is a medicinal composition for facilitating the growth, protection and healing of tissues and cells in animals and humans. The composition is formulated as a either a powder, gel, paste, film, fluid injectable, rehydratable freeze-dried paste or sponge, sprayable solution, topically applied patch with adhesive and reservoir system, an intermediate for coatables such as films and bandages, a matrix for membranes, or as a matrix of flexible polymer(s), or delivered as either an orally ingestible liquid, tablet or capsule. The main ingredient of the formulated compns. is hydrolyzed collagen, which can be combined with polysulfated glycosaminoglycans, hyaluronic acid or salts thereof, or a **glucosamine** salt, and mixts. thereof. The composition may be formulated as an aqueous eye drop solution

L5 ANSWER 11 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:453660 CAPLUS
DOCUMENT NUMBER: 143:13290
TITLE: Ultrasound assisted transdermal vaccine delivery method
INVENTOR(S): Cormier, Michel J. N.; Lin, Weiqi; Widera, Georg
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 27 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005112135	A1	20050526	US 2004-971338	20041021
AU 2004292953	A1	20050609	AU 2004-292953	20041021
CA 2546723	A1	20050609	CA 2004-2546723	20041021
WO 2005051455	A2	20050609	WO 2004-US35015	20041021
WO 2005051455	A3	20060413		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 1686904	A2	20060809	EP 2004-819508	20041021
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
CN 1905842	A	20070131	CN 2004-80040535	20041021
BR 2004016822	A	20070306	BR 2004-16822	20041021
JP 2007518468	T	20070712	JP 2006-541176	20041021
PRIORITY APPLN. INFO.:			US 2003-524062P P	20031121
			WO 2004-US35015 W	20041021

AB An apparatus and method for transdermally delivering a vaccine comprising a delivery system having (i) a microprojection member (or system) that includes a plurality of microprojections (or array thereof) that are adapted to pierce through the stratum corneum into the underlying epidermis layer, or epidermis and dermis layers and (ii) an ultrasonic device. In one embodiment, the vaccine is contained in a biocompatible coating that is applied to the microprojection member. In a further

embodiment, the delivery system includes a gel pack having a vaccine-containing hydrogel formulation that is disposed on the microprojection member after application to the skin of a patient. In an alternative embodiment, the vaccine is contained in both the coating and the hydrogel formulation. Thus, ultrasound can augment intracellular DNA uptake after delivery to skin by microprojection array or gel reservoir through microprojection array generated passages and can result in the induction of cellular and humoral immune responses to the antigen encoded by the delivered DNA vaccine construct.

L5 ANSWER 12 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:394533 CAPLUS
DOCUMENT NUMBER: 142:417213
TITLE: Spill resistant formulations containing clays
INVENTOR(S): Asotra, Satish; Wang, Xiaoli; Bodor, Zoltan
PATENT ASSIGNEE(S): Can.
SOURCE: U.S. Pat. Appl. Publ., 10 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005095263	A1	20050505	US 2004-975898	20041028
CA 2540869	A1	20050512	CA 2004-2540869	20041028
WO 2005041876	A2	20050512	WO 2004-US35851	20041028
WO 2005041876	A3	20050811		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1677755	A2	20060712	EP 2004-810086	20041028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				

PRIORITY APPLN. INFO.: US 2003-514999P P 20031028
WO 2004-US35851 W 20041028

AB The invention relates to novel spill resistant formulations comprising either a weak base or a weak acid as the pharmaceutical ingredient, a liquid base, a clay and a water soluble cellulose ether. The clay and cellulose ether allow for a broader pH range into which the pharmaceutically active agent may be dispersed or dissolved, and therefore allows for easier preparation and formulation of the pharmaceutical composition A spill resistant 200
mg/5 mL acyclovir suspension contained water 30.93, glycerin 50.0, polyethylene glycol-1000 15.0, Laponite 0.25, CM-cellulose 1.1 sucralose 0.5, and citric acid 0.4%. The initial viscosity of the suspension at 23° was cps 11000.

L5 ANSWER 13 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2005:244333 CAPLUS
DOCUMENT NUMBER: 143:307
TITLE: Atom, atom-type, and total nonstochastic and stochastic quadratic fingerprints: a promising

approach for modeling of antibacterial activity

AUTHOR(S): Marrero-Ponce, Yovani; Medina-Marrero, Ricardo; Torrens, Francisco; Martinez, Yamile; Romero-Zaldivar, Vicente; Castro, Eduardo A.

CORPORATE SOURCE: Department of Pharmacy, Faculty of Chemical-Pharmacy, Central University of Las Villas, Santa Clara, 54830, Cuba

SOURCE: Bioorganic & Medicinal Chemistry (2005), 13(8), 2881-2899
CODEN: BMECEP; ISSN: 0968-0896

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The Topol. Mol. Computer Design (TOMOCOMD-CARDD) approach has been introduced for the classification and design of antimicrobial agents using computer-aided mol. design. For this propose, atom, atom-type, and total quadratic indexes have been generalized to codify chemical structure information. In this sense, stochastic quadratic indexes have been introduced for the description of the mol. structure. These stochastic fingerprints are based on a simple model for the intramol. movement of all valence-bond electrons. In this work, a complete data set containing 1006 antimicrobial agents is collected and presented. Two structure-based antibacterial activity classification models have been generated. The models (including nonstochastic and stochastic indexes) classify correctly more than 90% of 1525 compds. in training sets. These models permit the correct classification of 92.28% and 89.31% of 505 compds. in an external test sets. The approach, also, satisfactorily compares with respect to nine of the most useful models for antimicrobial selection reported to date. Finally, a virtual screening of 87 new compds. reported in the anti-infective field with antibacterial activities is developed showing the ability of the models to identify new leads as antibacterial.

REFERENCE COUNT: 91 THERE ARE 91 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 14 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:531427 BIOSIS

DOCUMENT NUMBER: PREV200510324942

TITLE: Nutrient regulation of mTOR in the rat heart.

AUTHOR(S): Sharma, Saumya [Reprint Author]; Golfman, Leonard; Burgmaier, Mathias; Guthrie, Patrick; Chan, Suzanne; Haq, Syed; Taegtmeyer, Heinrich

CORPORATE SOURCE: Univ Texas, Houston Med Sch, Houston, TX 77030 USA

SOURCE: FASEB Journal, (MAR 4 2005) Vol. 19, No. 4, Suppl. S, Part 1, pp. A693.
Meeting Info.: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences. San Diego, CA, USA. March 31 -April 06, 2005. Amer Assoc Anatomists; Amer Assoc Immunologists; Amer Physiol Soc; Amer Soc Biochem & Mol Biol; Amer Soc Investigat Pathol; Amer Soc Nutr Sci; Amer Soc Pharmacol & Expt Therapeut; Int Union Physiol Sci.
CODEN: FAJOEC. ISSN: 0892-6638.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005

AB The mammalian target of rapamycin (mTOR), a kinase that serves as a convergence point for nutrient sensing and cell growth, is implicated in the developmentof cardiac hypertrophy. Because stimuli that induce hypertrophy also change myocardial metabolism, we postulated that

nutrients regulate mTOR in the heart.In the working rat heart, we investigated the role of glucose and amino acidsin regulating the mTOR pathway in response to three stimuli- insulin, **epinephrine**, and pressure overload. Furthermore, isolated cardiomyocytes were exposedto nutrient metabolites as well as activators/inhibitors of specific metabolic enzymes. Although insulin stimulated Akt phosphorylation, neither mTOR nor the downstream proteins, S6K1 and 4EBP1, were phosphorylated in the absence of glucose or amino acids. Only branched-chain amino acids and their metabolites induced S6K1 phosphorylation. **Glucosamine** and lactate, both metabolites of glucose, also activated mTOR. While iodoacetate, an inhibitor of glycolysis, decreased S6K1 phosphorylation, activation of AMP kinase with AICAR did not alter mTOR activity. Surprisingly, **epinephrine** and pressure overload induced the S6K1 phosphorylation in the absence of nutrients. However, depletion of glycogen prior to either stimulus attenuated S6K1 phosphorylation suggesting that endogenous nutrient stores also regulate mTOR activity. We conclude that hypertrophic stimuli activate mTOR in a nutrient-dependent manner indicating that metabolic and anabolic signals of the heart are linked. Specifically, branched-chainamino acid metabolism and glycolysis are required for the activation of mTOR by nutrients.

L5 ANSWER 15 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2004:780544 CAPLUS
DOCUMENT NUMBER: 141:301421
TITLE: Improved bioavailability and improved delivery of alkaline drugs
INVENTOR(S): Yu, Ruey J.; Van Scott, Eugene J.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004080468	A1	20040923	WO 2004-US6699	20040305
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004214215	A1	20041028	US 2004-792273	20040304
AU 2004220597	A1	20040923	AU 2004-220597	20040305
CA 2517782	A1	20040923	CA 2004-2517782	20040305
EP 1601366	A1	20051207	EP 2004-717955	20040305
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				

PRIORITY APPLN. INFO.:
US 2003-452557P P 20030307
US 2004-792273 A 20040304
WO 2004-US6699 A 20040305

OTHER SOURCE(S): MARPAT 141:301421

AB Embodiments of the invention relate to a composition, a process of making the composition, and to the use of the composition The compns. include a mol. complex

formed between an alkaline pharmaceutical and at least one selected from a hydroxyacid, a polyhydroxy acid, a related acid, a lactone, or combinations thereof. The compns. provide improved bioavailability and improved delivery of the drug into the cutaneous tissues. For example, diphenhydramine hydrochloride 29 g (0.1 mol) was dissolved in water (50 mL) and 5N sodium hydroxide (20 mL) was slowly added to generate diphenhydramine as a free base as shown by the formation of oily ppts. and the change from pH 5.5 to 9.4. Gluconolactone 18 g (0.1 mol) was added to form a mol. complex between the diphenhydramine free base and gluconic acid/gluconolactone as shown by the disappearance of the oily ppts. and the change from pH 9.4 to 7.4. The solution thus obtained contained 0.1 mol diphenhydramine in mol. complex with 0.1 mol gluconic acid/gluconolactone. This concentrated stock solution was used for various forms of topical formulations including oil-in-water creams, lotions, gels and solns.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 16 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:856942 CAPLUS
DOCUMENT NUMBER: 141:355338
TITLE: Oral administration form for veterinary use containing oils
INVENTOR(S): Yamin, Rina; First, Sigal; Rom, Alona
PATENT ASSIGNEE(S): CTS Chemical Industries Ltd., Israel
SOURCE: U.S. Pat. Appl. Publ., 5 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004202696	A1	20041014	US 2003-412260	20030414
AU 2004228786	A1	20041021	AU 2004-228786	20040325
CA 2522206	A1	20041021	CA 2004-2522206	20040325
WO 2004089337	A1	20041021	WO 2004-IL277	20040325
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1613279	A1	20060111	EP 2004-723296	20040325
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
JP 2006522804	T	20061005	JP 2006-507591	20040325

PRIORITY APPLN. INFO.: US 2003-412260 A 20030414
WO 2004-IL277 W 20040325

AB The present invention provides an oral administration form for animals, especially pets, comprising an oily liquid and at least one solid, oil-insol. active ingredient dispersed in said oily liquid The suspension is of a kind that n may be homogenized by shaking, and that may be easily poured out of a bottle. The oral administration form of the invention is preferably palatable to animals, it exhibits outstanding stability with water-sensitive active ingredients, and is readily accepted by animals

that has to take it. For example, a worm remover composition contained pyrantel pamoate 0.15, mineral oil 4.8 kg.

L5 ANSWER 17 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:995650 CAPLUS
DOCUMENT NUMBER: 141:416008
TITLE: Ion-pair delivery system for cosmetic and pharmaceutical compositions
INVENTOR(S): Gupta, Shyam K.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 8 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004228884	A1	20041118	US 2003-439349	20030515
US 2006147508	A1	20060706	US 2006-307729	20060218
US 2007092461	A1	20070426	US 2006-309441	20060806
PRIORITY APPLN. INFO.:			US 2002-265000	A2 20021004
			US 2002-280519	A2 20021025
			US 2002-290933	A2 20021107
			US 2003-394851	A2 20030322
			US 2003-439349	A2 20030515
			US 2006-307729	A2 20060218

AB This invention relates to a novel ion-pair delivery system useful for cosmetic, pharmaceutical, and topical nutraceutical applications in which the functional performance and consumer aesthetics of an electron donor composition and an electron acceptor composition, or a proton donor composition and a proton acceptor composition, are synergistically enhanced when such compns. are combined in an ion-pair mode. During ion-pair bonding process, the electron donor composition or the proton acceptor composition become pos. charged and the electron acceptor composition or proton donor composition become neg. charged and thus bind together in an ionic manner. Such ion-pair compns. release their electronically bound components in their original state when such compns. are absorbed into skin and reach physiol. pH conditions.

L5 ANSWER 18 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:293382 CAPLUS
DOCUMENT NUMBER: 140:309009
TITLE: Ascorbic acid salts of organic bases with enhanced bioavailability for synergistic anti-aging and skin protective cosmetic compositions
INVENTOR(S): Gupta, Shyam K.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004067890	A1	20040408	US 2002-265000	20021004
US 2006147508	A1	20060706	US 2006-307729	20060218
PRIORITY APPLN. INFO.:			US 2002-265000	A2 20021004

US 2002-280519	A2	20021025
US 2002-290933	A2	20021107
US 2003-394851	A2	20030322
US 2003-439349	A2	20030515

AB This invention relates to in-situ preparation, and stable topical delivery systems of ascorbic acid salts of organic bases that provide skin beneficial properties, including reduction in signs of skin aging, anti-wrinkle, anti-oxidant, and photo-protection from UV and sunlight. The formulation avoids the use of oils, minimizes the importance of the pH of the formulation, allows the incorporation of an aqueous solution of ascorbic acid or alkali metal salts of ascorbic acid in the formulation, does not require packaging the formulation in air tight containers, allows the use of large amts. of ascorbic acid, its salts, and its derivs., and does not require the use of expensive coatings. Moreover, several ascorbic acid derivs. of different chemical composition can be made in a stable topical formulation by the in-situ combination of readily available starting materials in a water solution, despite the understanding well known in the prior art that such compns. in water are inherently unstable. The in-situ method also permits the preparation of novel ascorbic acid derivs. that are not known in the prior art. For example, a topical composition for in-situ preparation of niacinamide ascorbate was prepared containing glycerin 43.3, methylparaben 0.2, ascorbic acid 9, niacinamide 6, deionized water 15, phenoxyethanol 0.9, Na cocoyl isethionate 20, Na Me cocoyl taurate 5, Actiplex 2789 0.1, and fragrance 0.5 part.

L5 ANSWER 19 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:269847 CAPLUS
DOCUMENT NUMBER: 140:297534
TITLE: Nitric oxide synthase inhibitor neuroprotective agents
INVENTOR(S): Yalpani, Manssur
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 27 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004063612	A1	20040401	US 2003-672257	20030926
WO 2004028548	A2	20040408	WO 2003-US30445	20030926
WO 2004028548	A3	20040826		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003272719	A1	20040419	AU 2003-272719	20030926

PRIORITY APPLN. INFO.:	US 2002-414694P	P	20020926
	WO 2003-US30445	W	20030926

AB The invention provides methods for treating neurodegenerative diseases with neuroprotective agents which inhibit nitric oxide synthase enzymes and in particular nitric oxide synthase III and can be used to treat Alzheimer's disease. Compds. of the invention include e.g. polyglutamate

polymers, and arabinogalactan compds.

L5 ANSWER 20 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:742234 CAPLUS
DOCUMENT NUMBER: 141:388909
TITLE: Selective Amine Recognition: Development of a Chemosensor for Dopamine and Norepinephrine
AUTHOR(S): Secor, Kristen E.; Glass, Timothy E.
CORPORATE SOURCE: Department of Chemistry, University of Missouri, Columbia, MO, 65211, USA
SOURCE: Organic Letters (2004), 6(21), 3727-3730
CODEN: ORLEF7; ISSN: 1523-7060
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 141:388909
AB A boronic acid-containing coumarin aldehyde was designed and synthesized. The sensor binds to catecholamines such as dopamine and norepinephrine by forming an iminium ion with the amine as well as a boronate ester with the catechol. An internal hydrogen bond produces a colorimetric response to these analytes with good selectivity for catecholamines over simple amines. The fluorescence of the sensor is quenched by the catechol.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 21 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:777540 CAPLUS
DOCUMENT NUMBER: 142:127075
TITLE: The Prophylactic Use of the β-Blocker Esmolol in Combination with Phosphodiesterase III Inhibitor Enoximone in Elderly Cardiac Surgery Patients
AUTHOR(S): Boldt, Joachim; Brosch, Christian; Lehmann, Andreas; Suttner, Stephan; Isgro, Frank
CORPORATE SOURCE: Department of Anesthesiology and Intensive Care Medicine, Klinikum der Stadt Ludwigshafen, Ludwigshafen, Germany
SOURCE: Anesthesia & Analgesia (Hagerstown, MD, United States) (2004), 99(4), 1009-1017
CODEN: AACRAT; ISSN: 0003-2999
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We assessed the influence of the prophylactic use of a combination of the IV β-adrenergic blocker, esmolol, and the phosphodiesterase III inhibitor, enoximone, on postbypass hemodynamic status, inflammation, and endothelial and organ function in a prospective, randomized, placebo-controlled study in 42 patients aged >65 yr undergoing aortocoronary bypass grafting. In 21 patients, esmolol (aim: heart rate <70 bpm) plus enoximone (initial bolus of 0.5 mg/kg followed by a continuous infusion of 2.5 μg · kg-1 · min-1) was started after induction of anesthesia and continued until the morning of the first postoperative day; another 21 patients received saline solution as placebo. Hemodynamics, splanchnic perfusion (gastric-arterial CO2 gap), liver function (glutathione transferase-α plasma levels), renal function (creatinine clearance, urine concns. of N-acetyl-β-D-**glucosaminidase**), myocardial ischemia (creatine-kinase MB and troponin T plasma levels), inflammation (elastase, interleukin-6 and -8 plasma levels), and endothelial integrity (adhesion mols. plasma levels) were assessed at baseline, before and after cardiopulmonary bypass (CPB), and in the intensive care unit until the first postoperative day. Catecholamine requirements were significantly less in the treated than in the nontreated patients. Heart rate was significantly slower, cardiac

index was higher, and gastric-arterial CO2 gap was significantly lower in the treatment group. Troponin T, β-N-acetyl-β-D-**glucosaminidase**, glutathione transferase-α, and soluble adhesion mols. increased significantly in the untreated control, but remained almost normal in the esmolol+enoximone patients. Inflammatory responses (elastase/interleukins) were attenuated by esmolol+enoximone. We conclude that, in comparison to an untreated control, the prophylactic use of a combination of esmolol and enoximone in elderly patients undergoing cardiac surgery with cardiopulmonary bypass resulted in overall beneficial effects on postbypass hemodynamic status, organ function, inflammatory response, and endothelial integrity.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 22 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:658504 CAPLUS
TITLE: Development of a chemosensor for dopamine
AUTHOR(S): Secor, Kristen E.; Glass, Timothy E.
CORPORATE SOURCE: Department of Chemistry, University of Missouri Columbia, Columbia, MO, 65211, USA
SOURCE: Abstracts of Papers, 228th ACS National Meeting, Philadelphia, PA, United States, August 22-26, 2004 (2004), ORGN-239. American Chemical Society: Washington, D. C.
CODEN: 69FTZ8
DOCUMENT TYPE: Conference; Meeting Abstract
LANGUAGE: English

AB Insufficient levels of the neurotransmitter dopamine are associated with Parkinson's disease, but the role of dopamine in the brain is not well understood. In an effort to gain insight into the function of dopamine in the brain, a chemosensor for dopamine has been developed. The sensor contains a boronic acid to bind the catechol of dopamine and a coumarin aldehyde which forms an imine with the amine portion. In a buffered aqueous solution at pH=7.0, the binding event is associated with a red shift in absorbance of 35 nm with KA=2.7*103 M-1. The binding constant for the related catecholamine norepinephrine is KA=1.8*103 M-1. The sensor does not respond to **epinephrine**, glucose, fructose, or cytidine. Other amine-containing analytes such as **glucosamine**, lysine, and glutamate bind with low affinity.

L5 ANSWER 23 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2004:273878 CAPLUS
DOCUMENT NUMBER: 141:271723
TITLE: Corticosteroids and β2-adrenergic agonists differentially modulate the synthesis and secretion of glycosaminoglycans by human lung cells
AUTHOR(S): Papakonstantinou, E.; Roth, M.; Tamm, M.; Karakiulakis, G.
CORPORATE SOURCE: Dept Pharmacology, School of Medicine, Aristotle University, Thessaloniki, 54124, Greece
SOURCE: Epitheorese Klinikes Farmakologias kai Farmakokinetikes, International Edition (2004), 18(1), 156-160
CODEN: EFKEEB; ISSN: 1011-6583
PUBLISHER: Pharmakon-Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Asthma is characterized by airway remodeling, involving changes in deposition of extracellular matrix mols. First-line therapy of persistent asthma involves the combination of inhaled corticosteroids and β2 adrenergic agonists. The aim of this study was to investigate the effect of corticosteroids, β2 agonists and their combination in the

secretion and deposition of glycosaminoglycans (GAGs) by human lung cells. Human bronchial epithelial and smooth muscle cells and lung fibroblasts were incubated for 48 h in the presence of budesonide, ciclesonide, **formoterol** or salmeterol. GAGs were determined in the cell culture medium and in the cell-associated matrix by 3H-**glucosamine** incorporation. We found that budesonide and ciclesonide resulted in a dose-dependent decrease in both cell-associated and secreted GAGs to approx. 50% of control levels. This effect was inhibited by the corticosteroid antagonist mifepristone, indicating the involvement of corticosteroid receptors. **Formoterol** and salmeterol had no effect. However, the combination of β2 agonists with corticosteroids further enhanced the inhibitory effect of corticosteroids. This effect was mediated via adrenergic receptors since it was abolished by propranolol. These results demonstrate that the anti-inflammatory action of corticosteroids when used alone or in combination with β2 adrenergic agonists in the treatment of asthma may also be associated with a beneficiary decrease in the deposition of matrix mols. in the lung.

REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 24 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4

ACCESSION NUMBER: 2004:556035 CAPLUS

DOCUMENT NUMBER: 141:241786

TITLE: Pro- and anti-inflammatory factors cooperate to control hyaluronan synthesis in lung fibroblasts

AUTHOR(S): Wilkinson, Thomas S.; Potter-Perigo, Susan; Tsoi, Christina; Altman, Leonard C.; Wight, Thomas N.

CORPORATE SOURCE: Department of Vascular Biology, Hope Heart Institute, Seattle, WA, USA

SOURCE: American Journal of Respiratory Cell and Molecular Biology (2004), 31(1), 92-99
CODEN: AJRBEL; ISSN: 1044-1549

PUBLISHER: American Thoracic Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Hyaluronan (HA) is an important constituent of the extracellular matrix and accumulates during inflammatory lung diseases like asthma. Little is known about the factors that regulate HA synthesis by lung cells. Accordingly, the authors investigated the effect of T-helper 1 (TH1) and 2 (TH2) cytokines and the anti-inflammatory agents fluticasone and **salmeterol** on HA synthesis in human lung fibroblasts. Interleukin-1β (IL-1β) and tumor necrosis factor (TNF)-α were the most potent stimulators of HA synthesis and when combined, caused synergistic increases in HA accumulation. Time-course anal. of HA accumulation and [3H]-**glucosamine** incorporation into HA demonstrated continued synthesis over the 24 h of stimulation. Peak synthesis at 6-12 h coincided with an increased proportion of high mol. weight HA. Reverse transcriptase polymerase chain reaction (RT-PCR) revealed that IL-1β and TNF-α induced HA synthase-2 mRNA 3 h following stimulation and remained elevated throughout the 24-h stimulation period. Fluticasone inhibited IL-1β and TNF-α induced HA synthesis (44.5%) whereas **salmeterol** had no effect. When combined, fluticasone and **salmeterol** inhibited HA synthesis to a greater extent (85.2%). Further, fluticasone attenuated IL-1β and TNF-α stimulated hyaluronan synthase-2 mRNA, and the addition of **salmeterol** cooperatively enhanced this inhibition. These results indicate that enhanced synthesis of HA by the proinflammatory cytokines IL-1β and TNF-α can be abrogated by specific corticosteroid and β2 blocker combinations shown to be effective in the treatment of asthma.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 25 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2006:80444 BIOSIS
DOCUMENT NUMBER: PREV200600076385
TITLE: Development of a chemosensor for dopamine.
AUTHOR(S): Secor, Kristen E. [Reprint Author]; Glass, Timothy E.
CORPORATE SOURCE: Univ Missouri, Dept Chem, Columbia, MO 65211 USA
SOURCE: Abstracts of Papers American Chemical Society, (AUG 22 2004) Vol. 228, No. Part 2, pp. U58.
Meeting Info.: Meeting of the Division of Chemical Toxicology of the American-Chemical-Society held at the 228th National Meeting of the American-Chemical-Society. Philadelphia, PA, USA. August 22 -26, 2004. Amer Chem Soc, Div Chem Toxicol.
CODEN: ACSRAL. ISSN: 0065-7727.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jan 2006
Last Updated on STN: 25 Jan 2006

L5 ANSWER 26 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 5

ACCESSION NUMBER: 2003:931192 CAPLUS
DOCUMENT NUMBER: 139:391355
TITLE: Combination of a beta-2 adrenoceptor agonists and an amino sugars and their use for the treatment of immunomodulatory disorders
INVENTOR(S): Weidner, Morten Sloth
PATENT ASSIGNEE(S): Astion Development A/s, Den.
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003097073	A1	20031127	WO 2003-DK263	20030422
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2482746	A1	20031127	CA 2003-2482746	20030422
AU 2003260279	A1	20031202	AU 2003-260279	20030422
EP 1496917	A1	20050119	EP 2003-752710	20030422
EP 1496917	B1	20051207		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 2005130935	A1	20050616	US 2003-512029	20030422
CN 1646143	A	20050727	CN 2003-808714	20030422
JP 2005529151	T	20050929	JP 2004-505071	20030422
AT 311889	T	20051215	AT 2003-752710	20030422
EP 1642574	A2	20060405	EP 2005-13726	20030422
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

NZ 536447	A	20060526	NZ 2003-536447		20030422
ES 2254960	T3	20060616	ES 2003-3752710		20030422
NO 2004005038	A	20041119	NO 2004-5038		20041119
HK 1073436	A1	20060303	HK 2005-105654		20050706
PRIORITY APPLN. INFO.:			DK 2002-586	A	20020419
			US 2002-373615P	P	20020419
			EP 2003-752710	A3	20030422
			WO 2003-DK263	W	20030422

AB The invention relates to combinations of an aminosugar and a beta-2 adrenoceptor agonist, such as salbutamol, for the treatment of diseases associated with hypersensitivity and inflammation, in particular hypersensitivity skin diseases. The aminosugar is preferably a monosaccharide derivative

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 27 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 6

ACCESSION NUMBER: 2003:511160 CAPLUS
DOCUMENT NUMBER: 139:63365
TITLE: Use of hyperforin or St. John's wort extracts for the treatment of anaphylactic shock and for maintaining and improving bone health
INVENTOR(S): Werz, Oliver; Albert, Dana; Steinhilber, Dieter; Bock, Andreas
PATENT ASSIGNEE(S): Phenion GMBH & Co. KG, Germany
SOURCE: PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2003053456	A1	20030703	WO 2002-EP14207		20021213
W: AU, BR, BY, CA, CN, DZ, HU, ID, IL, IN, JP, KR, MX, NO, NZ, PL, RO, RU, SG, UA, US, UZ, VN, YU, ZA					
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR					
DE 10163676	A1	20030710	DE 2001-10163676		20011221
AU 2002358698	A1	20030709	AU 2002-358698		20021213
PRIORITY APPLN. INFO.:			DE 2001-10163676	A	20011221
			WO 2002-EP14207	W	20021213

AB The invention discloses the use of hyperforin or St. John's wort (hypericum) exts. for the prophylaxis and/or therapy of anaphylactic shock and for maintaining and improving bone health, particularly for treating osteoporosis, and as a nutritional supplement and for pharmaceutical prepns. containing hyperforin or St John's wort extract

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 28 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:472344 CAPLUS
DOCUMENT NUMBER: 139:41818
TITLE: Preparation and therapeutic uses of compositions containing phenylalanine and leucine
INVENTOR(S): Ehrenpreis, Seymour; Howard, Lawrence
PATENT ASSIGNEE(S): Weller Health, Inc., USA
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003049687	A2	20030619	WO 2002-US38898	20021205
WO 2003049687	A3	20051222		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002359618	A1	20030623	AU 2002-359618	20021205
US 2004241256	A1	20041202	US 2004-497631	20040603
US 2006252727	A1	20061109	US 2006-479216	20060630
PRIORITY APPLN. INFO.:			US 2001-338320P	P 20011206
			WO 2002-US38898	W 20021205
			US 2004-497631	A1 20040603

AB The compns. of this invention comprise a mixture of (1) phenylalanine and a dietary food supplement, (2) leucine and a dietary food supplement, and (3) hydrocinnamic acid and a dietary food supplement. The compns. are used for medicinal purposes to alleviate a variety of maladies, including arthritis, anxiety, depression and inflammation. For example, the combination of D-phenylalanine (DPA) plus a NSAID, such as indomethacin or diclofenac, provided an enhanced degree of analgesia. The analgesia was far greater than the sum of the individual drugs. Similar results were obtained when DPA was combined with acetaminophen.

L5 ANSWER 29 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2002:429542 CAPLUS
DOCUMENT NUMBER: 137:11003
TITLE: Chondroprotective/restorative compositions containing hyaluronic acid
INVENTOR(S): Pierce, Scott W.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 14 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002068718	A1	20020606	US 2001-967977	20011002
US 6924273	B2	20050802		
US 2005182022	A1	20050818	US 2005-95632	20050401
PRIORITY APPLN. INFO.:			US 2000-237838P	P 20001003
			US 2001-967977	A1 20011002

AB An oral composition based on hyaluronic acid or its salts and optionally a therapeutic drug is provided for treating or preventing osteoarthritis, joint effusion, joint inflammation and pain, synovitis, lameness, post-operative arthroscopic surgery, deterioration of proper joint function including joint mobility, the reduction or inhibition of metabolic activity of chondrocytes, the activity of enzymes that degrade cartilage, and the reduction or inhibition of the production of hyaluronic acid in a mammal.

Addnl., compns. containing hyaluronic acid, chondroitin sulfate and **glucosamine** sulfate in a paste formulation are also described which can be administered on their own or can be used as a feed additive for cats and dogs. For example, a composition contained (by weight) **glucosamine** sulfate 36%, chondroitin sulfate 4%, sodium hyaluronate 0.144%, manganese sulfate 0.144%, ibuprofen 200 mg, powdered sugar 20%, glycerin 0.7%, xanthan gum 0.2%, sodium benzoate 0.7%, citric acid 0.2%, molasses 23.5%, and water 14.4%.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 30 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:10323 CAPLUS

DOCUMENT NUMBER: 136:74708

TITLE: Composition and method for the repair and regeneration of cartilage and other tissues based on a polymer gel

INVENTOR(S): Hoemann, Caroline D.; Buschmann, Michael D.; Mckee, Marc D.

PATENT ASSIGNEE(S): Biosyntech Canada Inc., Can.

SOURCE: PCT Int. Appl., 106 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002000272	A2	20020103	WO 2001-CA959	20010629
WO 2002000272	A3	20020808		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2412505	A1	20020103	CA 2001-2412505	20010629
US 2002082220	A1	20020627	US 2001-896912	20010629
EP 1294414	A2	20030326	EP 2001-947086	20010629
EP 1294414	B1	20060315		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004501682	T	20040122	JP 2002-505053	20010629
NZ 523763	A	20050225	NZ 2001-523763	20010629
AT 320277	T	20060415	AT 2001-947086	20010629
PT 1294414	T	20060731	PT 2001-947086	20010629
ES 2260241	T3	20061101	ES 2001-1947086	20010629
BR 2001012109	A	20070529	BR 2001-12109	20010629
MX 2003PA00203	A	20040913	MX 2003-PA203	20021219
IN 2003KN00072	A	20040814	IN 2003-KN72	20030120
ZA 2003000597	A	20040219	ZA 2003-597	20030122
HK 1055563	A1	20060526	HK 2003-106897	20030925
US 2006029578	A1	20060209	US 2005-31325	20050107
US 7148209	B2	20061212		
US 2007037737	A1	20070215	US 2006-584870	20061023
PRIORITY APPLN. INFO.:			US 2000-214717P	P 20000629
			US 2001-896912	B1 20010629
			WO 2001-CA959	W 20010629
			US 2005-31325	A1 20050107

AB The present invention relates to a new method for repairing human or animal tissues such as cartilage, meniscus, ligament, tendon, bone, skin, cornea, periodontal tissues, abscesses, resected tumors, and ulcers. The method comprises the step of introducing into the tissue a temperature-dependent polymer gel composition such that the composition adhere to the tissue and promote support for cell proliferation for repairing the tissue. Other than a polymer, the composition preferably comprises a blood component such as whole blood, processed blood, venous blood, arterial blood, blood from bone, blood from bone-marrow, bone marrow, umbilical cord blood, placenta blood, erythrocytes, leukocytes, monocytes, platelets, fibrinogen, thrombin and platelet rich plasma. The present invention also relates to a new composition to be used with the method of the present invention. For example, chondral defects with perforations to the subchrondal bone of rabbits were treated with a peripheral blood/chitosan-glyceryl phosphate mixture that was delivered as a liquid, and allowed to solidify in situ. After 5-8 wk healing, the blood/chitosan-treated defects were filled with repair tissue having the appearance of hyaline, a glycosaminoglycan (GAG)-rich cartilage repair tissue, which adhered to the defect surfaces, and filled the defects. Repair tissue from the untreated defects (control) had the appearance of fibro-cartilage, with particularly no metachromatic staining for GAG, and only partial defect filling.

L5 ANSWER 31 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:928122 CAPLUS
DOCUMENT NUMBER: 138:12504
TITLE: Method for assaying biomolecules and other constituents using indicator conjugates with synthetic nucleounits in lateral flow, liquid, and dry chemistry techniques
INVENTOR(S): Smith, Jack V.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 46 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002182600	A1	20021205	US 2001-829563	20010411
PRIORITY APPLN. INFO.:			US 2001-829563	20010411

AB The present invention is a method for the use of particles made up of nucleotides or fragments of base groups of DNA and RNA mols. herein referred to as synthetic nucleounits which can be used as recognition mols. with specificity and sensitivity significantly greater than that of antibodies which are used in clin. diagnostics, biotechnol., and research. The method for detecting an analyte using nucleounits targeted to the analyte comprises (1) identifying a nucleounit from a mixture of synthetic random sequences of nucleounit libraries, (2) conjugating the nucleounit to an indicator for the analyte, and (3) detecting the analyte using the nucleounit-indicator conjugate in a buffer. Step 1 is carried out by (a) contacting the analyte with the mixture of synthetic random sequences of nucleounit libraries such that some nucleounits bind the analyte, (b) removing the unbound nucleounits by partitioning, and (c) amplifying the remaining nucleounits by PCR to obtain an enriched solution of nucleounits with high affinity for the analyte. Thus, a method and lateral flow test strip for detection of cytomegalovirus (CMV) presence in a biol. sample such as serum or urine is described. The strip is prepared with three solns., one containing anti-CMV antibodies, one containing "nucleounit to CMV

antibody conjugated to red microparticles" and "red microparticles", and another containing "nucleounit to colored particles". The "nucleounit" may be an oligonucleotide aptamer specific for anti-CMV antibodies.

L5 ANSWER 32 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:736725 CAPLUS
DOCUMENT NUMBER: 137:268432
TITLE: Solid-dosage forms for weight loss product
INVENTOR(S): Fleischner, Albert M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 7 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002136782	A1	20020926	US 2001-761622	20010118
US 6420350	B1	20020716	US 2001-928715	20010813
PRIORITY APPLN. INFO.:			US 2001-761622 A2	20010118

AB Supplement compns. designed to support weight loss and increase energy are disclosed. The compns., to support weight loss and increase energy, comprise vitamin B6, zinc, manganese, chromium, Gymnema sylvestre leaf and extract, vanadium, **glucosamine** sulfate, lipotropic blend, appetite control blend, and thermogenic herbal concs. The compns. can be used in capsules or tablets.

L5 ANSWER 33 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:534030 CAPLUS
DOCUMENT NUMBER: 137:83682
TITLE: Weight loss products containing **glucosamine** and metabolic stimulants
INVENTOR(S): Fleischner, Albert M.
PATENT ASSIGNEE(S): Goen Group, Inc., USA
SOURCE: U.S., 4 pp., Cont.-in-part of U. S. Ser. No. 761,622.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6420350	B1	20020716	US 2001-928715	20010813
US 2002136782	A1	20020926	US 2001-761622	20010118
PRIORITY APPLN. INFO.:			US 2001-761622 A2	20010118

AB Disclosed are supplement compns. designed to support weight loss and increase energy. The composition of matter comprises **glucosamine** and a second component selected from the group consisting of caffeine and **ephedrine.** For example, a tablet contained vitamin B6 10 mg, chromium 20 μg, vanadium 20 μg, **glucosamine** sulfate 100 mg, Guarana seed and green tea exts. 910 mg (200 mg caffeine), and Ma huang exts. 312.5 mg (25 mg **ephedrine**) along with tablet excipients.
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 34 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:606371 CAPLUS
DOCUMENT NUMBER: 139:122720

TITLE: Formulation containing lysostaphin, its preparation and application
INVENTOR(S): Huang, Qingshan; Lu, Wanying
PATENT ASSIGNEE(S): Shanghai Gaoke Biological Engineering Co., Ltd., Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 10 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1367018	A	20020904	CN 2002-110672	20020128
CN 1438032	A	20030827	CN 2003-103476	20030127

PRIORITY APPLN. INFO.: CN 2002-110672 A 20020128

AB The composite preparation is composed of lysostaphin 0.1-20, lysozyme 50-85, bactericide (such as chlorhexidine or bacteriostatin) 1-20, salt (such as K, Na, and/or Mg salt of phosphate and/or chloride) 0.5-20, **glucosamine** derivative (such as chitosan, chitin, etc) 0.2-30, **ephedrine** 0-20, and antistaling agent (such as 4-hydroxybenzoic acid ester or Na benzoate) 0.1-20%. The composite preparation may be used as biol. bacteriocidal additive in external-used medical preparation, food, oral preparation, ear-use preparation, nose-use preparation, fish- specific preparation, and daily products for killing Neisseria gonorrhoeae, drug-resistant Staphylococcus aureus, Staphylococcus epidermidis, Candida albicans, Cyanobacteria, Escherichia coli, drug-resistant Micrococcus albicans, D-group enterococcus, Micrococcus tetragenus, Streptococcus anaerobius, etc.

L5 ANSWER 35 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:11688 CAPLUS
DOCUMENT NUMBER: 139:235516
TITLE: Enantioseparation of amino acids and drugs by CEC, pressure supported CEC, and micro-HPLC using a teicoplanin aglycone stationary phase
AUTHOR(S): Grobuschek, Nina; Schmid, Martin G.; Koidl, Julia; Gubitz, Gerald
CORPORATE SOURCE: Institute of Pharmaceutical Chemistry and Pharmaceutical Technology, Karl-Franzens University Graz, Graz, A-8010, Austria
SOURCE: Journal of Separation Science (2002), 25(15-17), 1297-1302
CODEN: JSSCCJ; ISSN: 1615-9306
PUBLISHER: Wiley-VCH Verlag GmbH & Co. KGaA
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Chiral separation of amino acids, drugs with amino alc. structure, and other drugs was investigated by capillary electrochromatog. (CEC), pressure supported CEC, and micro-HPLC on a teicoplanin aglycon stationary phase. The teicoplanin aglycon lacks the D-**glucosamine** and the D-mannose moieties and shows different separation behavior compared to a teicoplanin stationary phase. The selector is bonded to 3.5-μm silica gel and sepns. are carried out either in the reversed phase or the polar organic mode. This chiral stationary phase (CSP) shows high enantioselectivity especially for amino acids, e.g. for DOPA a Rs value of 10.5 was achieved. Although the chiral recognition ability for drugs is lower than that of intact teicoplanin, out of 22 β-blockers investigated, 16 are baseline separated by CEC.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 36 OF 106 MEDLINE on STN
ACCESSION NUMBER: 1999446182 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10516985
TITLE: Nutrition and dietary supplements.
AUTHOR: Fillmore C M; Bartoli L; Bach R; Park Y
CORPORATE SOURCE: Pendleton Community Care, Franklin, West Virginia, USA.
SOURCE: Physical medicine and rehabilitation clinics of North America, (1999 Aug) Vol. 10, No. 3, pp. 673-703. Ref: 177
Journal code: 9102787. ISSN: 1047-9651.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 11 Jan 2000
Last Updated on STN: 11 Jan 2000
Entered Medline: 16 Nov 1999

AB Quality and number of subjects in blinded controlled clinical trials about the nutrition and dietary supplements discussed here is variable. **Glucosamine** sulfate and chondroitin sulfate have sufficient controlled trials to warrant their use in osteoarthritis, having less side effects than currently used nonsteroidal anti-inflammatory drugs, and are the only treatment shown to prevent progression of the disease. Dietary supplements of **ephedrine** plus caffeine for weight loss (weight loss being the current first line recommendation of physicians for osteoporosis) show some promise, but are not sufficient in number of study subjects. Phenylpropanolamine is proven successful in weight loss. Both **ephedrine** and phenylpropanolamine have resulted in deaths and hence are worrisome [table: see text] as an over-the-counter dietary supplement. Other commonly used weight loss supplements like Cola acuminata, dwarf elder, Yohimbine, and Garcinia camborgia are either lacking controlled clinical trials, or in the case of the last two supplements, have clinical trials showing lack of effectiveness (although Garcinia has been successful in trials as part of a mixture with other substances, it is unclear if it was a necessary part of the mixture). Safety of these weight loss supplements is unknown. Chromium as a body building supplement for athletes appears to have no efficacy. Creatine may help more in weight lifting than sprinting, but insufficient study subjects and safety information make more studies necessary. Carbohydrate loading is used commonly before endurance competitions, but may be underused as it may be beneficial for other sport performances. Supplements for muscle injury or cramps have had too few studies to determine efficacy. Although proper rehydration with fluids and electrolytes is necessary, a paucity of actual studies to maximize prophylactic treatment for exercise induced cramping still exists. Nutritional supplements for cardiovascular disorders are generally geared to prevention. The United States Department of Agriculture has good recommendations to prevent atherosclerosis; a stricter version by Ornish was shown to reverse coronary heart disease, and the low meat, high fruit, and vegetable DASH diet has been found to decrease hypertension. The epidemiologic studies of hyperhomocysteinemia are impressive enough to give folic acid (or vitamin B6 or B12) supplements to those with elevated homocysteine levels and test patients who have a history of atherosclerotic disease, but no controlled clinical trials have been completed. Soluble fiber has several positive studies in reduction of cholesterol levels and generally is accepted. The data on vitamin E are the most confusing. This vitamin was not helpful in cerebrovascular prevention in China and not helpful at relatively small doses (50 mg) in the United States or Finland against major coronary events. Levels of 400 mg appeared to decrease cardiovascular disease in the United States in

studies based on reports by patients and in one large clinical trial. Vitamin E also was successful in prevention of restenosis after PTCA in one clinical trial. Both of these clinical trials need to be repeated in other developed country populations. Some nutritional and dietary supplements are justifiably useful at this point in time. Several meet the criteria of a late Phase 3 FDA clinical trial (where it would be released for public use), but many dietary supplements have insufficient numbers of studies. Some deaths also have occurred with some supplements. If these supplements were required to undergo clinical trials necessary for a new drug by the FDA, they would not be released yet to the public. Several nontoxic supplements appear promising, though need further study. Because they have essentially no toxicity (such as folic acid with B12, soluble fiber, and vitamin E) and may have efficacy, some of these supplementations may be useful now, without randomized clinical trials.

L5 ANSWER 37 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:16335 CAPLUS
DOCUMENT NUMBER: 128:57507
TITLE: Toward synthetic adrenaline receptors: strong, selective, and biomimetic recognition of biologically active amino alcohols by bisphosphonate receptor molecules
AUTHOR(S): Schrader, Thomas
CORPORATE SOURCE: Department of Organic Chemistry and Macromolecular Chemistry, Heinrich Heine-Universitaet Duesseldorf, Duesseldorf, D-40225, Germany
SOURCE: Journal of Organic Chemistry (1998), 63(2), 264-272
CODEN: JOCEAH; ISSN: 0022-3263
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Xylylene bisphosphonates represent a new class of artificial receptor mols. for alkylammonium ions. Mol. recognition takes place in a 1:1 chelate binding mode and an almost ideal array of short, linear hydrogen bonds is created, that guarantees maximum electrostatic and hydrogen bond interactions. The host mol. which was designed to imitate the natural adrenergic receptor is selective for 1,2- and 1,3-amino alcs. due to formation of an addnl. cooperative hydrogen bond between the phosphonate anion and the hydroxyl groups. Biol. important amino alcs. such as **glucosamine**, 1-aminosorbitol, **ephedrine** and the β-blocker propranol are bound in DMSO with Ka-values between 60000 and 130000 M-1. Secondary amines are complexed at least as strongly as their primary counterparts. The phosphonate ester groups allow lateral recognition of the substrate. This could be demonstrated for adrenaline model compds. which were recognized by phosphonates carrying extended aromatic ester groups for π,π-interactions.
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 38 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:224073 CAPLUS
DOCUMENT NUMBER: 126:216664
TITLE: Pharmaceutical compositions containing analgesics and antihistamines and methods for treating respiratory disorders
INVENTOR(S): Cramer, Ronald Dean; Mitra, Sekhar; Riker, Donald Kay
PATENT ASSIGNEE(S): Procter and Gamble Company, USA
SOURCE: PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9704808	A1	19970213	WO 1996-US12249		19960725
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE					
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA					
CA 2227958	A1	19970213	CA 1996-2227958		19960725
AU 9665991	A	19970226	AU 1996-65991		19960725
EP 841947	A1	19980520	EP 1996-925495		19960725
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI					
JP 11510168	T	19990907	JP 1996-507747		19960725
IN 1996DE01668	A	20050311	IN 1996-DE1668		19960726
ZA 9606385	A	19970604	ZA 1996-6385		19960728
PRIORITY APPLN. INFO.:			US 1995-508775	A	19950728
			US 1996-611528	A	19960305
			WO 1996-US12249	W	19960725

OTHER SOURCE(S): MARPAT 126:216664

AB Compns. and methods for providing improved treatment, management or mitigation of cold cold-like, allergy, sinus and/or flu symptoms by administering a safe and effective amount of a composition comprising an analgesic agent along with certain pyrrolidine and piperidine ether antihistaminic agents. A hard gelatin capsule contained ibuprofen 200.00, clemastine fumarate 0.67, pseudoephedrine.HCl 30.00 mg, and lactose q.s. Administration of 1-2 capsules every 4-12 h provide relief from cough, cold, flu and allergic rhinitis symptoms.

L5 ANSWER 39 OF 106 MEDLINE on STN DUPLICATE 8

ACCESSION NUMBER: 96370256 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8774152
TITLE: Dog pancreatic duct epithelial cells: long-term culture and characterization.
AUTHOR: Oda D; Savard C E; Nguyen T D; Eng L; Swenson E R; Lee S P
CORPORATE SOURCE: Department of Oral Biology, University of Washington, Seattle, USA.
SOURCE: The American journal of pathology, (1996 Mar) Vol. 148, No. 3, pp. 977-85.
Journal code: 0370502. ISSN: 0002-9440.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals.
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 8 Jan 1997

AB Epithelial cells, isolated from a normal dog pancreatic duct, were grown on collagen-coated culture inserts suspended above a feeder layer of myofibroblasts. The cells were examined by transmission electron microscopy, immunohistochemistry, cytogenetics, and flow cytometry. In addition, the constitutive and agonist-stimulated mucin secretion of these cells was studied using a [3H]N-acetyl-D-**glucosamine** labeling assay, and the stimulation of intracellular cAMP was measured. Cells grown on inserts with a feeder layer developed into confluent monolayers consisting of strictly polarized columnar epithelial cells with prominent microvilli, intercellular junctions, and normal chromosomal characteristics. They could be passaged repeatedly without a detectable

alteration in their morphology. The cells could also be grown on organotypic cultures, resulting in further differentiated cells simulating in vivo morphology. Immunohistochemistry demonstrated the presence of carbonic anhydrase II in these cells. Cells treated with vasoactive intestinal peptide, **epinephrine**, and dibutyryl-cAMP demonstrated a marked increase in mucin secretion compared with controls. In parallel experiments, VIP and **epinephrine** significantly increased intracellular cAMP. In conclusion we have developed a pancreatic epithelial cell preparation with morphology, cytokinetics, chromosomal, and DNA analyses characteristic of normal cells. Similar to normal columnar epithelial cells, these pancreatic duct cells secreted mucin constitutively and responded to agonist by increasing secretion via a cAMP-mediated pathway. They also contained carbonic anhydrase, which indicates that the cells are capable of secreting bicarbonate.

L5 ANSWER 40 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1995:546946 CAPLUS
DOCUMENT NUMBER: 122:274115
TITLE: Compositions containing an amino acid salt of a propionic acid nonsteroidal antiinflammatory agent and at least one of a decongestant, an expectorant, an antihistamine, and an antitussive
INVENTOR(S): Mitra, Sekhar
PATENT ASSIGNEE(S): Procter and Gamble Co., USA
SOURCE: PCT Int. Appl., 17 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9507103	A1	19950316	WO 1994-US9581	19940824
W: AU, BR, CA, CN, JP, PL, RU				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2170488	A1	19950316	CA 1994-2170488	19940824
AU 9476040	A	19950327	AU 1994-76040	19940824
EP 719156	A1	19960703	EP 1994-926020	19940824
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
CN 1130354	A	19960904	CN 1994-193312	19940824
BR 9407414	A	19961112	BR 1994-7414	19940824
JP 09502201	T	19970304	JP 1994-508695	19940824
PRIORITY APPLN. INFO.:			US 1993-116927 A	19930907
			WO 1994-US9581 W	19940824

AB A method for providing improved treatment, management, or mitigation of cold, coldlike, and/or flu symptoms comprises administering a safe and effective amount of a composition comprising certain amino acid salts of propionic acid nonsteroidal antiinflammatory agents along with ≥1 of a decongestant, expectorant, antihistamine, and antitussive. Thus, a hard gelatin capsule contained naproxen lysinate 200, pseudoephedrine-HCl 30, astemizole 5, and glyceryl guaiacolate 100 mg.

L5 ANSWER 41 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1995:662901 CAPLUS
DOCUMENT NUMBER: 123:65866
TITLE: Liquid ophthalmic sustained release delivery system
INVENTOR(S): Yen, Shau Fong; Reed, Kenneth W.
PATENT ASSIGNEE(S): Ciba-Geigy Corporation, USA
SOURCE: U.S., 8 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5422116	A	19950606	US 1994-198924	19940218
CA 2181715	A1	19950824	CA 1995-2181715	19950209
CA 2181715	C	20060530		
WO 9522315	A1	19950824	WO 1995-IB82	19950209
W: AM, AU, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, JP, KG, KP, KR, KZ, LK, LR, LT, LV, MD, MG, MN, MX, NO, NZ, PL, RO, RU, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9514649	A	19950904	AU 1995-14649	19950209
AU 704353	B2	19990422		
EP 744938	A1	19961204	EP 1995-906463	19950209
EP 744938	B1	20010905		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
CN 1140988	A	19970122	CN 1995-191655	19950209
JP 09509166	T	19970916	JP 1995-521696	19950209
JP 3720359	B2	20051124		
AT 205079	T	20010915	AT 1995-906463	19950209
PT 744938	T	20011228	PT 1995-906463	19950209
ES 2163495	T3	20020201	ES 1995-906463	19950209
IL 112674	A	19991222	IL 1995-112674	19950216
ZA 9501323	A	19950818	ZA 1995-1323	19950217
FI 9603217	A	19960816	FI 1996-3217	19960816
FI 117851	B1	20070330		
NO 9603431	A	19961009	NO 1996-3431	19960816
NO 313861	B1	20021216		
GR 3036884	T3	20020131	GR 2001-401754	20011016
PRIORITY APPLN. INFO.:			US 1994-198924 A	19940218
			WO 1995-IB82 W	19950209

AB Disclosed is a sustained release liquid aqueous ophthalmic delivery system and a method of providing a slow and sustained release of ophthalmic treating agents to the eye of a mammal which comprises administering to the eye of a said mammal an effective amount of a homogeneous liquid aqueous ophthalmic pharmaceutical composition, of pH between about 3.0 and about 6.2, which is administrable in drop form and which comprises an ophthalmically effective concentration of a said ophthalmic treating agent and about 0.05% to about 10% by weight of the polymer chitosan; said polymer consisting essentially of (A) monomeric β(1→4)-D- **glucosamine** linked units and of (B) monomeric β(1→4)-D- **glucosamine** linked units which are scattered randomly in the mol. of the polymer, the numerical proportions of A and B being from about 60 to about 99% of A and about 1 to about 40% of B, the viscosity rating being from about 3 to about 3000 cP; in which method, upon contact with the higher pH of the ocular fluid, said liquid formulation is converted to a stiff gel from which the ophthalmic treating agent is slowly released over a prolonged period of time.

L5 ANSWER 42 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 9
ACCESSION NUMBER: 1995:296460 CAPLUS
DOCUMENT NUMBER: 122:47293
TITLE: PGE generates intracellular cAMP and accelerates mucin secretion by cultured dog gallbladder epithelial cells
AUTHOR(S): Kuver, R.; Savard, C.; Oda, D.; Lee, S. P.

CORPORATE SOURCE: Dep. Med. Oral Biol., Univ. Washington Sch. Med., Seattle, WA, 98108, USA
SOURCE: American Journal of Physiology (1994), 267(6, Pt. 1), G998-G1003
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mucin is the main secretory product of gallbladder epithelial cells. Increased gallbladder mucus secretion has been implicated in gallstone formation in humans. The mechanisms underlying control of mucin synthesis and secretion by the gallbladder are not known. This study aimed to elucidate the efficacy of a panel of secretagogues to stimulate mucin secretion and to determine the intracellular second messengers involved. Studies were carried out on normal well-differentiated epithelial cells from dog gallbladder grown in monolayer culture. Intracellular cAMP as measured by RIA increased in response to prostaglandin (PG) E2, PGE1, vasoactive intestinal peptide, **epinephrine**, and isoproterenol. The greatest effect, a 37-fold increase in cAMP level, was noted with PGE2 at 1.0 μM concentration In contrast, three breakdown products of phosphatidylinositol (inositol triphosphate, inositol bisphosphate and inositol monophosphate) were not detected with any of the secretagogues tested. Assay of mucin secretion using tritiated N-acetyl-D-**glucosamine**, a mucin precursor, showed that the same secretagogues noted to increase intracellular cAMP led to an increase in mucin secretion. No correlation was noted, however, between the magnitude of the intracellular cAMP rise and the amount of mucin secreted. A membrane-permeable form of cAMP, dibutyryl cAMP, mimicked PGE2-induced mucin secretion. The results unequivocally show that secretagogue-stimulated mucin secretion in these normal gallbladder epithelial cells can proceed via a cAMP signal transduction pathway.

L5 ANSWER 43 OF 106 MEDLINE on STN
ACCESSION NUMBER: 95116108 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7816422
TITLE: Effects of antiglaucoma agents on glycosaminoglycans in organ-cultured rabbit trabecular meshwork.
AUTHOR: Yoneyama J
CORPORATE SOURCE: Department of Ophthalmology, Shinshu University School of Medicine, Matsumoto, Japan.
SOURCE: Ophthalmologica. Journal international d'ophtalmologie. International journal of ophthalmology. Zeitschrift fur Augenheilkunde, (1994) Vol. 208, No. 5, pp. 278-83.
Journal code: 0054655. ISSN: 0030-3755.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 17 Feb 1995
Last Updated on STN: 17 Feb 1995
Entered Medline: 6 Feb 1995

AB The effect of antiglaucoma drugs on glycosaminoglycans (GAGs) of the rabbit trabecular meshwork (TM) was studied by organ culture and sequential enzymatic degradation (SED). Whole TM explants from one rabbit eye were dissected into 8 parts, then divided into control, **epinephrine**-, timolol- and pilocarpine-treated groups. After 2 weeks of culture, and incubation with medium containing radiolabeled precursors for 48 h, GAGs of the TM were isolated and subjected to SED. Treatment with **epinephrine** significantly reduced the hyaluronic acid and increased the chondroitin sulfate in radiolabeled precursor incorporation rate ($p < 0.001$). Other drugs had little effect. Thus,

epinephrine appeared to influence the biosynthesis of GAGs of rabbit TM.

L5 ANSWER 44 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1994:182763 CAPLUS
DOCUMENT NUMBER: 120:182763
TITLE: Effect of **procaterol** on human inflammatory cells
AUTHOR(S): Kobayashi, Nobuko; Kobayashi, Hideki; Baker, Amanda J.; Fuller, Richard W.
CORPORATE SOURCE: R. Postgrad. Med. Sch., Univ. London, Du, London, W12 ONN, UK
SOURCE: Showa University Journal of Medical Sciences (1993), 5(1), 1-11
CODEN: SUMSEG; ISSN: 0915-6380
DOCUMENT TYPE: Journal
LANGUAGE: English

AB β-Adrenergic agonists are used in the treatment of bronchial asthma for their bronchodilator effect, in addition, they may have anti-allergic effects. The authors investigated the effects of **procaterol**, a long acting β2 agonist, on human inflammatory cells including monocytes, neutrophils, basophils and lung mast cells. **Procaterol** caused dose-related inhibition of thromboxane B2 (TXB2) release from monocytes; this effect was completely abolished by propranolol, suggesting that it is mediated via β2 receptors. In contrast, there was no inhibition of N-acetyl-β-D- **glucosaminidase** (NAG) release. Further, **procaterol** dose-dependently inhibited superoxide anion release from neutrophils, which was partially reduced by propranolol. **Procaterol** had no effect on formyl-Methionyl-Leucyl-Phenylalanine (FMLP)-induced neutrophil chemotaxis. **Procaterol** dose-dependently inhibited lung mast cell histamine release after anti-IgE challenge; this effect was completely abolished by pre-treatment with propranolol. **Procaterol** (10-4 M) only inhibited basophil histamine release 5 min after anti-IgE stimulation. Thus, **procaterol**, has inhibitory effects on some but not all functions of human monocytes, neutrophils, basophils, and mast cells.

L5 ANSWER 45 OF 106 MEDLINE on STN
ACCESSION NUMBER: 91281810 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2058654
TITLE: Rat sublingual gland as a model to study glandular mucous cell secretion.
AUTHOR: Culp D J; Graham L A; Latchney L R; Hand A R
CORPORATE SOURCE: Department of Dental Research, University of Rochester, New York 14642.
CONTRACT NUMBER: P50-DE-07003 (NIDCR)
R01-HL-32949 (NHLBI)
SOURCE: The American journal of physiology, (1991 Jun) Vol. 260, No. 6 Pt 1, pp. C1233-44.
Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 18 Aug 1991
Last Updated on STN: 3 Mar 2000
Entered Medline: 30 Jul 1991

AB To study the regulation of mucous cell secretion, we have developed an in vitro cell model consisting of enzymatically dispersed mucous acinar

structures (cell aggregates) from rat sublingual glands. Histological and ultrastructural evidence demonstrates that the cell aggregates are highly enriched in mucous cells, retain the morphological and ultrastructural features observed in intact glands, and undergo transition to an extensive secretory state when stimulated by 10 microM carbachol. The secretory responsiveness of the cell aggregates was verified in pharmacological studies. Carbachol stimulated secretion in a dose-dependent manner with high affinity (concentration causing half-maximal response = 0.3 microM) and was completely inhibited by atropine. Secretion was also stimulated by vasoactive intestinal peptide and substance P but not by alpha- or beta-adrenergic agonists. Biochemical characterization of secretion during nonstimulated and carbachol-stimulated conditions (after preincubation in [3H]**glucosamine**) demonstrated that, in response to carbachol, cell aggregates synthesized and secreted mucins which were similar to mucin glycoproteins isolated from whole glands. Collectively, our results establish that the rat sublingual cell aggregate model is a viable and pharmacologically responsive cell system to study the regulation of mucous cell secretion.

L5 ANSWER 46 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 1991:401582 CAPLUS
DOCUMENT NUMBER: 115:1582
TITLE: Adrenergic interaction in mucin secretion of rat submandibular gland in vitro
AUTHOR(S): Taylor, Samuel E.; McWhorter, Kathleen
CORPORATE SOURCE: Dep. Pharmacol., Baylor Coll. Dent., Dallas, TX, 75246, USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics (1991), 257(2), 582-6
CODEN: JPETAB; ISSN: 0022-3565
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Maximally effective concns. of the mixed α and β adrenoceptor agonists **epinephrine** and norepinephrine cause greater amts. of mucin secretion than the pure β adrenoceptor agonist isoproterenol, and this response requires extracellular calcium. The nature of the effect of the putative pure α adrenoceptor agonist phenylephrine on isoproterenol-induced mucin secretion was examined, as was the role of extracellular calcium in this interaction. A graphical method for determining whether interaction between 2 drugs is additive, antagonistic, or synergistic was used, to analyze this interaction. Submandibular glands were removed from male rats, divided into sections, and placed in modified Hank's balanced salt solution Mucin secretion was measured as acid precipitable disintegrations per min after labeling the submandibular gland with [14C]**glucosamine.** Phenylephrine caused a small secretion of mucin, which was blocked partially by phentolamine and completely by propranolol. Phenylephrine caused a shift of the isoproterenol concentration-response curve to the left of the theor. curve expected if 2 drugs act additively. Mucin secretion induced by isoproterenol alone was independent of extracellular calcium concentration; however, the combination of isoproterenol and phenylephrine caused a concentration-dependency on extracellular calcium.

L5 ANSWER 47 OF 106 MEDLINE on STN DUPLICATE 11
ACCESSION NUMBER: 92101483 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1759395
TITLE: [Lysosomal leukocyte glycosidases in childhood bronchial asthma].
Lizosomnye glikozidazy leikotsitov pri bronkhial'noi astme u detei.
AUTHOR: Gerasimov A A; Balabolkin I I; Bakanov M I
SOURCE: Voprosy medit sinskoi khimii, (1991 Sep-Oct) Vol. 37, No.

5, pp. 35-8.
Journal code: 0416601. ISSN: 0042-8809.
PUB. COUNTRY: USSR
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 23 Feb 1992
Last Updated on STN: 23 Feb 1992
Entered Medline: 4 Feb 1992

AB Activity of lysosomal glycosidases beta-galactosidase, N-acetyl-beta-D-galactosaminidase and N-acetyl-beta-D-**glucosaminidase** was studied in lymphocytes and neutrophils of children with bronchial asthma. Alterations of the enzymatic activity were found to depend considerably on the disease stage, form, severity, duration and on the sensitization efficiency. Activity of these enzymes was markedly decreased after glucocorticoid treatment while **clenbuterol** did not affect their activity. The status of leukocyte lysosomal glycosidases appears to be of importance for development of bronchial asthma in children.

L5 ANSWER 48 OF 106 MEDLINE on STN
ACCESSION NUMBER: 91357144 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1653152
TITLE: [The efficacy of **glucosamine** in treating complicated hyperreactive experimental myocardial infarct]. Effektivnost' gliukozamina v lechenii oslozhnennogo giperreaktovnogo eksperimental'nogo infarkta miokarda.
AUTHOR: Sokrut V N; Zupanets I A; Iabluchanskii N I; Drogovoz S M
SOURCE: Farmakologiia i toksikologiia, (1991 Mar-Apr) Vol. 54, No. 2, pp. 31-3.
Journal code: 16920420R. ISSN: 0014-8318.
PUB. COUNTRY: USSR
DOCUMENT TYPE: (COMPARATIVE STUDY)
(ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 27 Oct 1991
Last Updated on STN: 27 Oct 1991
Entered Medline: 9 Oct 1991

AB The effect of **glucosamine** hydrochloride on the course of complicated hyperreactive myocardial infarction in dogs was studied. **Glucosamine** contributed to the restoration of reactivity in the animals. The reactivity became corresponding for normoreactive myocardial infarction. **Glucosamine** normalized carbohydrate and protein metabolism in the necrosis zone, cAMP and cGMP and also their ratio that led to healing by postinfarction scar, and in none of the cases the heart aneurysm developed. The optimizing healing of hyperreactive myocardial infarction under the influence of **glucosamine** is mediated through the mechanisms of reactivity and the regulation of metabolic processes.

L5 ANSWER 49 OF 106 MEDLINE on STN
ACCESSION NUMBER: 90225423 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2158256
TITLE: Direct inhibitory action of glucocorticoid on glycoconjugate secretion from airway submucosal glands.
AUTHOR: Shimura S; Sasaki T; Ikeda K; Yamauchi K; Sasaki H; Takishima T

CORPORATE SOURCE: First Department of Internal Medicine, Tohoku University School of Medicine, Sendai, Japan.
SOURCE: The American review of respiratory disease, (1990 Apr) Vol. 141, No. 4 Pt 1, pp. 1044-9.
Journal code: 0370523. ISSN: 0003-0805.
PUB. COUNTRY: United States
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199005
ENTRY DATE: Entered STN: 22 Jun 1990
Last Updated on STN: 29 Jan 1999
Entered Medline: 17 May 1990

AB The precise mechanism by which glucocorticoids inhibit airway mucus secretion is still unknown. To study directly the effect of glucocorticoid on submucosal gland secretion, we examined the effects of dexamethasone on the precursor uptake, biosynthesis, and release of mucus glycoprotein in isolated feline tracheal submucosal glands. Mucus glycoprotein release from isolated glands was estimated by measuring [3H] **glucosamine**-labeled trichloroacetic acid (TCA)-precipitable glycoconjugates secreted into the medium. Released glycoconjugate per hour per dry weight of gland tissue was less than 7% of the total intracellular content, where intracellular content is defined as total 3H activity in the dissolved gland tissue. Treatment with 10(-9) to 10(-5) M dexamethasone for 24 to 72 h significantly reduced basal glycoconjugate secretion up to 22% of control (a 78% decrease) in a dose-dependent fashion, whereas the total intracellular 3H content was reduced to 70% of control (a 30% decrease) with no statistically significant differences from controls. The ratio of released glycoconjugates to the total intracellular content decreased significantly to 31% of control (a 69% decrease) after the treatment with 10(-10) to 10(-5) M dexamethasone. Further, ratio of radioactivity of TCA-precipitable glycoconjugates in the dissolved gland tissue to the total intracellular 3H content increased from 40% in nontreated controls to 46% after the treatment with dexamethasone (10(-5) M). Dexamethasone also inhibited the glycoconjugate secretion stimulated by dibutyryl cyclic AMP and alpha- and beta-adrenergic agonists. Simultaneously, the ratio of released to total intracellular content also decreased significantly after dexamethasone treatment. (ABSTRACT TRUNCATED AT 250 WORDS)

L5 ANSWER 50 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1991:223259 CAPLUS
DOCUMENT NUMBER: 114:223259
TITLE: Significant differences in the structural basis of the induction of sister chromatid exchanges and chromosomal aberrations in Chinese hamster ovary cells
AUTHOR(S): Rosenkranz, Herbert S.; Ennever, Fanny K.; Dimayuga, Mario; Klopman, Gilles
CORPORATE SOURCE: Dep. Environ. Health Sci., Case West. Reserve Univ., Cleveland, OH, USA
SOURCE: Environmental and Molecular Mutagenesis (1990), 16(3), 149-77
CODEN: EMMUEG; ISSN: 0893-6692
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The structural basis of the induction of sister chromatid exchanges (SCE) and chromosomal aberrations (Cvt) in Chinese hamster ovary cells was investigated by the CASE (Computer Automated Structure Evaluation) method. Using the relevant National Toxicol. Program data bases, CASE identified a set of structural determinants responsible for the induction of SCE and

another one for Cvt. A comparison between the structural determinants associated with SCE and Cvt revealed an overlap of only 22.6%, while the overlap between SCE and the determinants of mutagenicity in Salmonella is 54.5%. Apparently, the structural bases of the two phenomena differ; it is likely that SCE, but not Cvt, involves a significant electrophilic/DNA-damaging component.

L5 ANSWER 51 OF 106 MEDLINE on STN DUPLICATE 12
ACCESSION NUMBER: 91054691 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2242093
TITLE: Effect of centrifugal force and catecholamines on glycosaminoglycans synthesis of vascular smooth muscle cells in culture.
AUTHOR: Hamada M; Kusuyama Y; Nishio I; Ura M; Takeda J; Hano T; Masuyama Y
CORPORATE SOURCE: Department of Medicine, Wakayama Medical College, Japan.
SOURCE: Atherosclerosis, (1990 Aug) Vol. 83, No. 2-3, pp. 147-53. Journal code: 0242543. ISSN: 0021-9150.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 8 Feb 1991
Last Updated on STN: 8 Feb 1991
Entered Medline: 21 Dec 1990

AB To evaluate the effect of hypertension on glycosaminoglycan (GAG) synthesis, cultured vascular smooth muscle cells (CVSMCs) from the aorta of spontaneously hypertensive rats (SHR) and Wistar-Kyoto rats (WKY) were exposed to centrifugal forces and catecholamines. GAG synthesis of CVSMCs was measured by the incorporation of [3H]**glucosamine** into GAGs which were secreted into the culture medium for 24 h. Basal level of GAG synthesis was much higher in SHR than in WKY, when expressed in terms of DNA contents. When exposed to centrifugal force, CVSMCs from rats of both strains synthesized more GAGs. GAG synthesis was enhanced by both noradrenaline (NA) and adrenaline (Ad) in WKY. The enhanced GAG synthesis in WKY by NA or Ad was prevented by pretreatment with propranolol, but not prazosin. In SHR, NA and Ad did not enhance GAG synthesis at this concentration of catecholamines. However, the effects of propranolol or prazosin on GAG synthesis in SHR, when incubated with either NA or Ad, were compatible with the phenomena observed in WKY. Adding dibutyryl cyclic AMP to the culture medium enhanced GAG synthesis in rats of both strains. These data suggest that not only the mechanical stress of high intra-arterial pressure but also beta receptor stimulation, via increasing cyclic AMP, enhance GAG synthesis of vascular smooth muscle cells in hypertension.

L5 ANSWER 52 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1989:608457 CAPLUS
DOCUMENT NUMBER: 111:208457
TITLE: 1,2-Bis(4-methoxyphenyl)ethylenediamine as a fluorogenic reagent for reducing carbohydrates
AUTHOR(S): Umegae, Yoshihiko; Nohta, Hitoshi; Ohkura, Yosuke
CORPORATE SOURCE: Fac. Pharm. Sci., Kyushu Univ., Fukuoka, 812, Japan
SOURCE: Analytica Chimica Acta (1989), 217(2), 263-70
CODEN: ACACAM; ISSN: 0003-2670
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Eight 1,2-diarylethylenediamines in the meso- or DL-form produced fluorescence when heated with reducing carbohydrates in an alkaline medium. Of the diamines, meso-1,2-bis(4-methoxyphenyl)ethylenediamine is the most

favorable reagent for reducing carbohydrates, including 2-deoxy sugars, amino sugars and sialic acids. The reagent permits the fluorometric determination of reducing carbohydrates at concns. as low as 0.2-0.9 nmol mL-1.

L5 ANSWER 53 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1989:151950 CAPLUS
DOCUMENT NUMBER: 110:151950
TITLE: Effects of lectins with various carbohydrate binding specificities on lipid metabolism in isolated rat and hamster adipocytes
AUTHOR(S): Ng, T. B.; Li, W. W.; Yeung, H. W.
CORPORATE SOURCE: Dep. Biochem., Chin. Univ. Hong Kong, Shatin, Hong Kong
SOURCE: International Journal of Biochemistry (1989), 21(2), 149-55
CODEN: IJBOBV; ISSN: 0020-711X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mannose-binding and N-acetylglucosamine-binding lectins exhibited potent antilipolytic and lipogenic activities. Fucose-binding lectins had minimal lipogenic activity but possessed antilipolytic activity. Most galactose-binding and N-acetylgalactosamine-binding lectins were devoid of significant antilipolytic and lipogenic activities. Notable exceptions were lectins from Momordica charantia, Wisteria floribunda, Vicia villosa, Codium fragile, and the Siberian pine tree. Bauhinia purpurea lectin was exceptional in that it exhibited potent lipogenic activity but lacked antilipolytic activity. The galactose-binding horse gram and osage orange lectins exerted antilipolytic activity in hamster but not in rat adipocytes.

L5 ANSWER 54 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1989:128652 CAPLUS
DOCUMENT NUMBER: 110:128652
TITLE: Method for accelerated wound healing by administration of anti-inflammatory agents in combination with other active ingredients
INVENTOR(S): Meisner, Lorraine F.
PATENT ASSIGNEE(S): Peritain Ltd., USA
SOURCE: U.S., 9 pp. Cont.-in-part of U.S. 4,647,453.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 4772591	A	19880920	US 1986-852522	19860416
US 4590067	A	19860520	US 1984-662069	19841018
US 4647453	A	19870303	US 1985-778811	19850925
AU 8548702	A	19860424	AU 1985-48702	19851015
AU 573160	B2	19880526		
CA 1261263	A1	19890926	CA 1985-493238	19851017
JP 61097221	A	19860515	JP 1985-231403	19851018
JP 06051626	B	19940706		

PRIORITY APPLN. INFO.: US 1984-662069 A2 19841018
US 1985-778811 A2 19850925

AB The healing of wounds caused by trauma or surgery is accelerated by administration of (1) ascorbic acid 1 - 58, (2) biol. available Ca 2 - 10, (3) a precursor or stimulant of **epinephrine** or norepinephrine 0.5-2.5, and (4) an anti-inflammatory substance 1-5 parts by weight A powder

contained bone meal 4.6, **glucosamine** 2.8, ascorbic acid 3.2, and tyrosine 1.0 part by weight. Patients having periodontal diseases brushed their teeth twice daily using the above powder and the pocket depth was measured periodically; the powder was effective in the treatment of periodontal disease, either to decrease inflammation prior to surgery or, in some cases, to even replace surgery.

L5 ANSWER 55 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 13
ACCESSION NUMBER: 1989:51279 CAPLUS
DOCUMENT NUMBER: 110:51279
TITLE: Effect of drugs in vitro on lysosomal enzyme activities in bovine retinal pigment epithelial cells
AUTHOR(S): Hayasaka, Seiji; Noda, Sachiko; Setogawa, Tomoichi
CORPORATE SOURCE: Dep. Ophthalmol., Shimane Med. Univ., Izumo, 693, Japan
SOURCE: Japanese Journal of Ophthalmology (1988), 32(3), 316-21
CODEN: JJOPA7; ISSN: 0021-5155
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of several drugs on the activities of lysosomal enzymes were studied in the crude extract of bovine retinal pigment epithelial cells. Acid phosphatase, β-D-glucuronidase, N-acetyl-β-D-**glucosaminidase**, α-L-fucosidase, and α-D-mannosidase were studied as lysosomal enzymes. Sodium iodate at 10-6, 10-5, and 10-4M, and potassium iodate at 10-5 and 10-4M inhibited acid phosphatase activity. Ferrous chloride at 10-5 and 10-4M suppressed β-D-glucuronidase activity. Ferric chloride, indomethacin, chloroquine, chlopromazine, 5-fluorouracil, tobramycin, daunomycin, oxalate, and **epinephrine** had no or only minimal inhibitory effects on the lysosomal enzyme activities examined

L5 ANSWER 56 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1988:280997 BIOSIS
DOCUMENT NUMBER: PREV198835009311; BR35:9311
TITLE: INFLUENCE OF **EPINEPHRINE** ON GLYCOSAMINOGLYCAN SYNTHESIS IN CALF TRABECULAR MESHWORK CELL CULTURE.
AUTHOR(S): RODMAN K D [Reprint author]; RICHARDSON T M
CORPORATE SOURCE: MASS EYE EAR INFIRMARY, HARV MED SCH, BOSTON, MASS, USA
SOURCE: Investigative Ophthalmology and Visual Science, (1988) Vol. 29, No. ABSTR. ISSUE, pp. 86.
Meeting Info.: ANNUAL SPRING MEETING OF THE ASSOCIATION FOR RESEARCH IN VISION AND OPHTHALMOLOGY, SARASOTA, FLORIDA, USA, MAY 1-6, 1988. INVEST OPHTHALMOL VISUAL SCI.
CODEN: IOVSDA. ISSN: 0146-0404.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 7 Jun 1988
Last Updated on STN: 7 Jun 1988

L5 ANSWER 57 OF 106 MEDLINE on STN
ACCESSION NUMBER: 88275964 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2839764
TITLE: Use of 1-deoxymannojirimycin to show that complex oligosaccharides regulate cellular distribution of the alpha 1-adrenergic receptor glycoprotein in BC3H1 muscle cells.
AUTHOR: Terman B I; Insel P A
CORPORATE SOURCE: Department of Pharmacology, University of California, San Diego, La Jolla 92093.

CONTRACT NUMBER: GM 31987 (NIGMS)
HL 35018 (NHLBI)
HL 35847 (NHLBI)
SOURCE: Molecular pharmacology, (1988 Jul) Vol. 34, No. 1, pp. 8-14.
Journal code: 0035623. ISSN: 0026-895X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198808
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 17 Aug 1988

AB We have previously shown that alpha 1-adrenergic receptors in BC3H1 muscle cells are glycoproteins containing complex but not high mannose oligosaccharides. In the present study we investigated the role of the complex sugars in functional aspects of the receptor by treating BC3H1 cells with 1-deoxymannojirimycin (dMM), which blocks conversion of high mannose oligosaccharides to complex chains. Receptors were photoaffinity labeled in intact cells with 125I-azido prazosin; drug treatment with dMM resulted in conversion of the 87-kDa receptor to 62 kDa. The 62-kDa protein was sensitive to mannosidase, indicating loss of complex sugars. Radioligand ([3H]prazosin) binding analysis carried out at 37 degrees to intact cells indicated that dMM treatment increased the affinity of the alpha 1-receptors for [3H]prazosin 2-fold and decreased the number of total cellular receptors by 15%. In order to distinguish between surface and sequestered receptors, we assessed [3H]prazosin binding to intact cells at 4 degrees using competition by the hydrophilic agonist **epinephrine** to define surface receptors and by nonradioactive antagonists (prazosin and phentolamine) to define total receptors. In control cells, **epinephrine** competed for 90% of the total receptors, whereas for dMM-treated cells this value was only 60%. In addition, dMM treatment caused a 40% reduction in **epinephrine** -stimulated phosphatidylinositol turnover when compared with untreated cells. The results indicate that dMM treatment reduces the number of functional alpha 1-adrenergic receptors on the cell surface while increasing the number of sequestered receptors. We conclude that complex oligosaccharides are important for cellular localization and function of alpha 1-adrenergic receptors in BC3H1 cells.

L5 ANSWER 58 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 14

ACCESSION NUMBER: 1988:35295 CAPLUS
DOCUMENT NUMBER: 108:35295
TITLE: Relations between glycoprotein synthesis and carbohydrate metabolism in small intestinal mucosa: effect of cholera enterotoxin
AUTHOR(S): Vengrov, P. R.; Cherkasova, I. D.; Yurkiv, V. A.
CORPORATE SOURCE: Cent. Res. Inst. Epidemiol., Moscow, USSR
SOURCE: Biokhimiya (Moscow) (1987), 52(11), 1914-18
CODEN: BIOHAO; ISSN: 0006-307X
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB The regulatory properties of adenylate cyclase in small intestinal mucosa were investigated. Glucagon, **epinephrine**, and isoproterenol failed to activate the cAMP synthesis; prostaglandin E1 caused a 2.8-fold activation and cholera toxin produced a 4.5-fold stimulation. The latter was not able to increase the rate of glucose synthesis from alanine in vitro, but increased markedly the in vivo incorporation of 14C-labeled alanine into the mucus **glucosamine.** Unlabeled

glucosamine excretion was also enhanced 3-fold. This provides evidence for the involvement of glycolytic and gluconeogenetic enzyme systems in mucosal glycoprotein synthesis. It was assumed that both metabolic pathways may play a common physiol. role, namely, to convert carbohydrates and gluconeogenic precursors into the substrate for **glucosamine** synthesis which is thought to be a rate-limiting step in small intestinal mucus secretion.

L5 ANSWER 59 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 15
ACCESSION NUMBER: 1987:96626 CAPLUS
DOCUMENT NUMBER: 106:96626
TITLE: Insulin-stimulated diacylglycerol production results from the hydrolysis of a novel phosphatidylinositol glycan
AUTHOR(S): Saltiel, Alan R.; Sherline, Peter; Fox, Judith A.
CORPORATE SOURCE: Lab. Biochem. Endocrinol., Rockefeller Univ., New York, NY, 10021, USA
SOURCE: Journal of Biological Chemistry (1987), 262(3), 1116-21
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The insulin [9004-10-8]-dependent release of a carbohydrate substance from plasma membranes which regulated certain intracellular enzymes was recently described. This enzyme-modulating substance appeared to arise from the phosphodiesterase hydrolysis of a novel inositol-containing glycolipid. This is supported by observations that insulin stimulated the rapid generation of [3H]myristate-labeled diacylglycerol in cultured BC3H1 myocytes. Myristoyl diacylglycerol production in these cells was unaffected by **epinephrine**, although arachidonate-labeled diacylglycerol was rapidly produced in response to stimulation by this α1-adrenergic agent. The production of distinct species of diacylglycerol was apparently due to hormonally specific hydrolysis of different precursors. A novel glycolipid was identified on silica TLC or HPLC which served as a substrate for the insulin-stimulated phosphodiesterase reaction. This glycolipid was metabolically labeled with radioactive inositol, **glucosamine**, and myristic acid, which suggestes a phosphatidylinositol (PI)-glycan structure. Treatment of this glycolipid with a PI-specific phospholipase C [9001-86-9] resulted in the generation of 2 products: an inositol phosphate-glycan which modulated the activity of the low-Km cAMP phosphodiesterase and myristoyl diacylglycerol. Insulin caused the rapid hydrolysis of the PI-glycan, which was then apparently resynthesized. Thus, insulin stimulates the activity of a phospholipase C which selectively hydrolyzes a novel PI-glycan, releasing a carbohydrate enzyme modulator as well as a unique species of diacylglycerol.

L5 ANSWER 60 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 16
ACCESSION NUMBER: 1987:629866 CAPLUS
DOCUMENT NUMBER: 107:229866
TITLE: **Clenbuterol**-induced muscle growth: investigation of possible mediation by insulin
AUTHOR(S): McElligott, Mary Ann; Mulder, Jean E.; Chaung, Lee Yuh; Barreto, Albert, Jr.
CORPORATE SOURCE: Dep. Anim. Drug Discovery, Merck Sharp and Dohme Res. Lab., Rahway, NJ, 07065, USA
SOURCE: American Journal of Physiology (1987), 253(4, Pt. 1), E370-E375
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The role of insulin as a possible mediator of the β-adrenergic

agonist stimulation of muscle growth was investigated. To exclude possible action of the β-agonist on the pancreatic release of insulin, diabetes was induced in rats by a streptozotocin injection (100 mg/kg). Insulin levels were almost not detectable in these rats. Feeding either normal diet or diet containing the β-adrenergic agonist **clenbuterol** (10 parts/million) did not alter plasma insulin concns. The effects of **clenbuterol** on muscle and weight gain were determined in diabetic rats given daily insulin replacement (D + I) and fed either a normal diet or **clenbuterol**-treated diet. **Clenbuterol**, fed for 1 wk, increased the wet weight of the gastrocnemius, soleus, and extensor digitorum longus muscles (15-23%) in both normal and D + I rats. Although **clenbuterol** increased body weight gain, it did not alter feed consumption and, therefore, feed efficiency (g gain/g food) was improved. Activities of cathepsin B and N-acetyl-β- **glucosaminidase**, but not cathepsin D, were elevated in the soleus muscles of **clenbuterol**-treated rats. The **clenbuterol**-induced increase in muscle growth in the insulin-replaced diabetic rats indicated that this β-adrenergic agonist effect was not mediated by an alteration of circulating levels of insulin, secondary to β-agonist action on pancreatic insulin release.

L5 ANSWER 61 OF 106 MEDLINE on STN DUPLICATE 17
ACCESSION NUMBER: 87025969 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2876709
TITLE: Heterogeneity between soluble human and rabbit splenic alpha 2-adrenoceptors.
AUTHOR: McKernan R M; Dickinson K E; Miles C M; Sever P S
SOURCE: Biochemical pharmacology, (1986 Oct 15) Vol. 35, No. 20, pp. 3517-23.
Journal code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198611
ENTRY DATE: Entered STN: 2 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 19 Nov 1986

AB The pharmacological and biochemical characteristics of soluble alpha 2-adrenoceptors were investigated to determine whether differences observed in membranes were maintained in solution and to probe the nature of any such differences. alpha 2-Adrenoceptors were solubilized from purified plasma membrane preparations of human and rabbit spleen using digitonin. [3H]yohimbine bound to one population of alpha 2-adrenoceptors in the preparations with dissociation constants of 2.4 nM and 7.8 nM respectively. The pharmacological profile of the alpha 2-adrenoceptors has been examined. Upon solubilization the affinity of the alpha 2-adrenoceptors for yohimbine was unchanged. In contrast, the potency of idazoxan and RX 811066 were increased, whereas the potency for prazosin (human only), phentolamine and WY 26392 was decreased 2-3-fold. The potency of the agonists oxymetazoline, UK 14304 and adrenaline were all reduced upon solubilization of alpha 2-adrenoceptors. The selectivity of yohimbine, idazoxan, RX 811066 and WY 26392 for human rather than rabbit alpha 2-adrenoceptors was maintained in solution. Possible sources of heterogeneity between human and rabbit alpha 2-adrenoceptors were investigated. The protein structure was probed by comparing the susceptibility of the receptors to inactivation by sulphydryl modifying agents. No differences were observed in the potency of N-ethylmaleimide or p-chloromercuribenzoate to inactivate the receptor. The carbohydrate component of the receptors was investigated using agarose-linked lectins. Rabbit splenic alpha 2-adrenoceptors had a lower affinity for the lectins

wheatgerm agglutinin (Triticum vulgaris) and soybean (Glycine max) which bind the sugars N-acetyl d-**glucosamine** and N-acetyl d-galactosamine respectively. These findings suggest that heterogeneity of the alpha 2-adrenoceptor derives from its structural characteristics rather than its environment in the membrane.

L5 ANSWER 62 OF 106 MEDLINE on STN
ACCESSION NUMBER: 87177072 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3031785
TITLE: Effect of **fenoterol** on secretions of an isolated single submucosal gland from the trachea.
AUTHOR: Sasaki H; Sasaki T; Shimura S; Takishima T
SOURCE: Respiration; international review of thoracic diseases, (1986) Vol. 50 Suppl 2, pp. 266-9.
Journal code: 0137356. ISSN: 0025-7931.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198705
ENTRY DATE: Entered STN: 3 Mar 1990
Last Updated on STN: 29 Jan 1999
Entered Medline: 19 May 1987
AB The mechanism of secretion of airway submucosal glands is not known. Although the existence of cyclic AMP in the submucosal gland has been reported, the effect of stimulation of the gland by exogenous cyclic AMP has been denied. We hypothesize that the negative effect of exogenous cyclic AMP is due to the modified influence of ciliated epithelium on the submucosal gland, because epithelium releases chemical mediators. We isolated a single submucosal gland from a cat trachea. We measured mucous glycoprotein labeled with [3H]-**glucosamine** from the isolated submucosal glands. With stimulation of dibutyryl cyclic AMP, a percent increase of the secretion above control was about 3 times in isolated glands. Methacholine and **fenoterol** secreted mucus largely in isolated glands. We suggest that cyclic AMP increased the secretion in the glands. The difference of quantity of secretions between methacholine and **fenoterol** has been investigated further.

L5 ANSWER 63 OF 106 MEDLINE on STN
ACCESSION NUMBER: 85305477 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3929669
TITLE: [Intermediate lobe of the amphibian pituitary gland: an endocrine gland with multiple secretions and under multi-hormonal control].
Le lobe intermediaire de l'hypophyse des amphibiens: une glande endocrine a secretions multiples et sous controle pluri-hormonal.
AUTHOR: Tonon M C; Leroux P; Jenks B G; Gouteux L; Jegou S; Guy J; Pelletier G; Vaudry H
SOURCE: Annales d'endocrinologie, (1985) Vol. 46, No. 2, pp. 69-87.
Ref: 126
Journal code: 0116744. ISSN: 0003-4266.
PUB. COUNTRY: France
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198510
ENTRY DATE: Entered STN: 20 Mar 1990

Last Updated on STN: 20 Mar 1990
Entered Medline: 22 Oct 1985

AB The cells of the frog pars intermedia synthesize a 36 000 (36K) protein called proopiomelanocortin (POMC). After [3H]**glucosamine** incorporation, separation of newly synthesized products by SDS-polyacrylamide gel electrophoresis showed that this 36K protein was glycosylated. Tryptic mapping revealed only one site of glycosylation and showed that the carbohydrate side-chain was located in the N-terminal region of POMC. The 36K protein was not released by the melanotrophs, but it generated, through specific intracellular proteolytic cleavage, a number of smaller peptides which were subsequently released. These peptides were identified by various methods including selective amino-acid incorporation, HPLC purification, acid-urea gel electrophoresis, tryptic and chymotryptic mapping, assay of melanotropic activity, radioimmunoassays and immunoprecipitations. Some of the newly synthesized N-terminal (18K) fragment of the POMC was secreted intact while a portion of it was further processed, via an intermediate peptide, to give mature gamma-MSH. All three of these peptides were glycosylated. In addition, the mature peptide (gamma-MSH) exhibited a low but significant melanotropic activity. The C-terminal portion of the prohormone was very rapidly processed to give des N alpha-acetyl alpha-MSH, corticotropin-like-intermediate lobe peptide (CLIP) and beta-endorphin. Authentic alpha-MSH was always absent in cellular extracts: acetylation to give rise to alpha-MSH was a late enzymatic process strictly linked to hormonal release. Since acetylation of alpha-MSH is required for full biological activity of this peptide, it is possible to conceive that this later step could be under neuroendocrine control. Using the perifusion technique we have been able to show the complexity of the control mechanisms regulating amphibian melanotrophs. It is generally accepted that the aminergic innervation of the intermediate lobe of the pituitary is involved in the hypothalamic control of melanotropin release. We have demonstrated that, in amphibians, dopamine inhibits alpha-MSH secretion through D2-type dopaminergic receptors whereas norepinephrine and (or) **epinephrine** stimulate alpha-MSH secretion via beta-adrenergic receptors. The existence of peptidergic fibers within parenchymal cells of the pars intermedia has been demonstrated. Evidence for TRH-containing fibers has been obtained by immunohistochemistry. Using a specific radioimmunoassay for TRH, we have confirmed the presence of TRH in the neurointermediate lobe of the frog. We have shown that TRH is a powerful MSH-releasing factor in these animals. (ABSTRACT TRUNCATED AT 400 WORDS)

L5 ANSWER 64 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 18
ACCESSION NUMBER: 1984:469928 CAPLUS
DOCUMENT NUMBER: 101:69928
TITLE: Regulation of the synthesis of mucin glycoproteins in swine trachea explants
AUTHOR(S): Lloyd, Cindy; Kennedy, John R.; Mendicino, Joseph
CORPORATE SOURCE: Dep. Biochem., Univ. Georgia, Athens, GA, 30602, USA
SOURCE: In Vitro (1984), 20(5), 416-32
CODEN: ITCSAF; ISSN: 0073-5655
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Swine tracheal epithelium was cultured as explants in a chemical defined medium for ≤2 wks. Explants viability was shown by ultrastructure preservation and by incorporation of radioactive **glucosamine** and SO42- into secreted mucin glycoproteins. The rate of secretion of mucin glycoprotein was apprx.0.035 mg/cm2/day. An initial 24 h lag period was due to the equilibration of intracellular mucin glycoprotein pools with radioactive precursors. The rate of glycoprotein secretion showed a linear dependence on explant area. Maximal incorporation was 200 μM **glucosamine.** A higher concentration of 35SO42-, 1000 μM, was required for maximal incorporation of the precursor. Insulin at 0.1-1 μg/mL

increased the rate of secretion 2-fold, whereas 0.1-100 μg hydrocortisone/mL and 0.1-100 μg **epinephrine**/mL significantly decreased the rate of secretion. Vitamin A had little or no effect on normal trachea explants at low concns., and, at higher concns., 10-5 M, it decreased the secretion of mucin glycoproteins. Vitamin A, a 10-9 M, increased the rate of synthesis of glycoprotein ≥4-fold in trachea explants from vitamin A-deficient rats. Mucus secretions collected from the surface of swine trachea and from the culture medium of trachea explants were purified. The mucus was solubilized by reduction and carboxymethylation, and the high-mol.-weight mucin glycoproteins were purified by chromatog. on Sepharose CL-6B columns under dissociating conditions in 2M guanidine-HCl. The mucin glycoproteins purified from swine trachea and from the culture medium of trachea explants were virtually indistinguishable. They showed the same properties when examined by gel electrophoresis and immunopptn. The purified glycoproteins contained .apprx.25% protein, and serine, threonine, and proline were the principal amino acids present. More than 80% of the oligosaccharide chains in both samples were released by treatment with alkaline borohydride. Nearly the same molar ratio of N-acetylgalactosamine, N-acetylglucosamine, galactose, fucose, sulfate, and sialic acid was found in both prepns.

L5 ANSWER 65 OF 106 MEDLINE on STN
ACCESSION NUMBER: 85118711 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6240928
TITLE: Effects of the H2-receptor agonist dimaprit on lymphocyte responsiveness in vitro.
AUTHOR: Binderup L
SOURCE: Agents and actions, (1984 Oct) Vol. 15, No. 3-4, pp. 119-24.
Journal code: 0213341. ISSN: 0065-4299.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198503
ENTRY DATE: Entered STN: 20 Mar 1990
Last Updated on STN: 20 Mar 1990
Entered Medline: 5 Mar 1985
AB The histamine H2-agonist dimaprit was found to increase the response of rat spleen cells to the T-cell mitogen Concanavalin A, when present at concentrations of 10(-5) and 10(-4)M. Higher concentrations of dimaprit were cytotoxic. The enhanced response seemed to be associated with an inhibitory effect of dimaprit on T-suppressor cell activity rather than with a direct mitogen-like stimulation of lymphocyte proliferation or with an interference with monocyte/macrophage functions. The stimulatory effects of dimaprit were not reversed by the H2-receptor antagonist, cimetidine, nor by the beta-receptor antagonists metoprolol and H 35/25. Addition of the H1-receptor antagonist, mepyramine, further increased the stimulatory effect of dimaprit on lymphocyte responsiveness.

L5 ANSWER 66 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1984:158311 BIOSIS
DOCUMENT NUMBER: PREV198427074803; BR27:74803
TITLE: ALPHA-1 ADRENERGIC RECEPTOR AND BETA-2 ADRENERGIC RECEPTORS CO EXPRESSED ON CLONED MDCK CELLS ARE DISTINCT GLYCO PROTEINS.
AUTHOR(S): MEIER K E [Reprint author]; STERNFELD D R; INSEL P A
CORPORATE SOURCE: DIV PHARMACOL M-013-H, DEP MED, UNIV CALIF, SAN DIEGO, LA JOLLA, CALIF 92093, USA
SOURCE: Biochemical and Biophysical Research Communications, (1984)

Vol. 118, No. 1, pp. 73-81.
CODEN: BBRCA9. ISSN: 0006-291X.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: ENGLISH

L5 ANSWER 67 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1984:83952 BIOSIS
DOCUMENT NUMBER: PREV198427000444; BR27:444
TITLE: INFLUENCE OF AMINES ON PLATELET FUNCTION.
AUTHOR(S): GERARD J M [Reprint author]; HOUSTON D S; MCCREA J M; GLOVER S; BUTLER A M
CORPORATE SOURCE: MANITOBA INST OF CELL BIOL, WINNIPEG, MANITOBA, CANADA
SOURCE: Thrombosis and Haemostasis, (1983) Vol. 50, No. 1, pp. 289. Meeting Info.: 9TH INTERNATIONAL CONGRESS ON THROMBOSIS AND HEMOSTASIS, JULY 4-8, 1983. THROMB HEMOSTASIS.
CODEN: THHADQ. ISSN: 0340-6245.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH

L5 ANSWER 68 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1984:204210 CAPLUS
DOCUMENT NUMBER: 100:204210
TITLE: Hormonal regulation of some regulatory enzymes in the testis of rat
AUTHOR(S): Reddy, P. R. K.; Madhubala, R.; Rukmini, V.
CORPORATE SOURCE: Sch. Life Sci., Univ. Hyderabad, Hyderabad, 500 134, India
SOURCE: Proc. Symp. Cell. Control Mech. (1983), Meeting Date 1982, 83-9. Dep. At. Energy: Bombay, India.
CODEN: 51GBA3
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Prostaglandins (PGE2 [363-24-6] and PGF2α [551-11-1]) and catecholamines (**epinephrine** [51-43-4] and norepinephrine [51-41-2]) stimulated ornithine decarboxylase (EC 4.1.1.17) (ODC) [9024-60-6] activity in both Leydig cells and seminiferous tubules of immature rats, whereas the ODC-stimulating activity of LH [9002-67-9] is restricted to Leydig cells and that of FSH [9002-68-0] to seminiferous tubules. Addnl. stimulation of ODC activity was observed after norepinephrine, LH, FSH, or PGE2 administration to animals pretreated with **epinephrine.** Exposure of rat testis to PGE2 for 24 h desensitized the testis to a 2nd dose of PGE2, FSH, LH, or cAMP [60-92-4]. **Glucosamine** 6-phosphate synthase (EC 5.3.1.19) [9030-45-9] activity of the testis was stimulated by FSH and pregnant mare serum gonadotropin [9002-70-4] but was unaffected by LH, LH-RH [9034-40-6], and human chorionic gonadotropin [9002-61-3]. Evidently, ODC and **glucosamine** 6-phosphate synthase are specifically regulated by various hormones in the testis of rats.

L5 ANSWER 69 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1982:290591 BIOSIS
DOCUMENT NUMBER: PREV198274063071; BA74:63071
TITLE: EFFECTS OF THE CALMODULIN ANTAGONIST TRIFLUOPERAZINE ON STIMULUS INDUCED CALCIUM MOBILIZATION AGGREGATION SECRETION AND PROTEIN PHOSPHORYLATION IN PLATELETS.
AUTHOR(S): FEINSTEIN M B [Reprint author]; HADJIAN R A
CORPORATE SOURCE: DEP OF PHARMACOL, UNIV OF CONNECTICUT HEALTH CENTER, FARMINGTON, CONN 06032, USA

SOURCE: Molecular Pharmacology, (1982) Vol. 21, No. 2, pp. 422-431.
CODEN: MOPMA3. ISSN: 0026-895X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The calmodulin antagonist trifluoperazine inhibited human platelet aggregation and the secretion of Ca2+ and ATP from dense granules and N-acetylglucosaminidase from lysosomes. The inhibition of aggregation and secretion occurred concurrently but could also be demonstrated independently of each other and in the absence of any effect on stimulus-induced platelet shape change. All platelet-stimulating agents tested were antagonized, i.e., thrombin, trypsin, collagen, **epinephrine**, A23187 [calcimycin] melittin, thimerosal and arachidonic acid. Trifluoperazine can block thrombin-induced release of arachidonic acid from phosphatidyl choline and thromboxane formation without inhibiting breakdown of phosphatidyl inositol or the formation of phosphatidic acid. Despite a lack of effect of trifluoperazine on the metabolism of exogenous arachidonic acid, both arachidone acid- and prostaglandin H2-induced aggregation and secretion were antagonized. Trifluoperazine could also completely suppress thrombin-induced phosphorylation of platelet proteins, notably the MW 20,000 myosin L chain and a MW 47,000 protein. The effect on the latter may be due to inhibition of the Ca2+-dependent, phospholipid-activated, protein kinase C, whereas the effects on myosin phosphorylation can be attributed to inhibition of calmodulin-dependent myosin L chain kinase. Similarities in the process of protein kinase C and calmodulin-dependent enzymes may account for their common susceptibility to the same class of inhibitors. Using chlortetracycline as a fluorescent probe for intracellular membrane-bound Ca2+ showed that platelet stimulation releases Ca2+ intracellularly prior to secretion and protein phosphorylation. Trifluoperazine acted to uncouple activation from secretion: at low concentrations the drug inhibited secretion, although intracellular Ca2+ release was either not affected or actually markedly speeded up by the drug. Higher concentrations of trifluoperazine partially inhibited later stages of Ca2+ release and almost totally blocked secretion. Evidently, calmodulin is involved in mediating certain effects of Ca2+ in platelets, but other calmodulin-independent processes are also targets for trifluoperazine.

L5 ANSWER 70 OF 106 MEDLINE on STN DUPLICATE 19
ACCESSION NUMBER: 83101515 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6817815
TITLE: Effects of N-acetylglucosamine and platelet inhibitors on the synergistic interaction of platelets and aggregating agents in the presence of wheat germ agglutinin.
AUTHOR: Tandon N N; Ordinas A; Jamieson G A
CONTRACT NUMBER: HL 209071 (NHLBI)
SOURCE: Biochimica et biophysica acta, (1982 Nov 24) Vol. 719, No. 2, pp. 388-95.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198303
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 11 Mar 1983

AB Low concentrations of wheat germ agglutinin (4 micrograms/ml) have been shown to act synergistically to induce platelet aggregation with **epinephrine**, collagen, arachidonate and ionophore A23187.

Aggregation ceased on the addition of the haptenic sugar N-acetylglucosamine at any time following the onset of aggregation with these agonists and a small degree of disaggregation was observed during the reversible first wave with the biphasic aggregating agents **epinephrine** and ADP. Cyclooxygenase inhibitors such as indomethacin and aspirin blocked the second wave of aggregation with the biphasic aggregating agents **epinephrine** and ADP but a synergistic response continued to be shown with the first wave in the presence of these inhibitors. Release of [14C]serotonin and the mobilization of [3H]arachidonate by **epinephrine** and collagen were markedly stimulated in the presence of wheat germ agglutinin but there was no increase of either radiolabel in the case of ADP. Platelet shape change, but not aggregation, occurred with low levels of wheat germ agglutinin and the synergistic response with ADP, collagen or ionophore A23187 occurred without further shape change. Wheat germ agglutinin did not affect the basal or stimulated levels of cyclic AMP. The membrane fluidity of platelets was not affected by the lectin or by thrombin as shown by the lack of change in fluorescence polarization with diphenylhexatriene. It is suggested that the binding of wheat germ agglutinin to the platelet surface induces platelet activation by mechanisms similar to those of other agonists and that it may affect the distribution of membrane-bound Ca2+ by a reversible perturbation of the platelet membrane.

L5 ANSWER 71 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1983:180724 BIOSIS
DOCUMENT NUMBER: PREV198375030724; BA75:30724
TITLE: CIRCADIAN RHYTHM IN URINARY N ACETYL-BETA **GLUCOSAMINIDASE** EC-2.3.1.30 OF CLINICALLY HEALTHY SUBJECTS TIMING AND PHASE RELATION TO OTHER URINARY CIRCADIAN RHYTHMS.
AUTHOR(S): LAKATUA D J [Reprint author]; BLOMQUIST C H; HAUS E; SACKETT-LUNDEEN L; BERG H; SWOYER J
CORPORATE SOURCE: DEP ANATOMIC AND CLIN PATHOLOGY, ST PAUL-RAMSEY MED CENTER, 640 JACKSON ST, ST PAUL, MINN 55101, USA
SOURCE: American Journal of Clinical Pathology, (1982) Vol. 78, No. 1, pp. 69-77.
CODEN: AJCPAI. ISSN: 0002-9173.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Urinary N-acetyl-β- **glucosaminidase** (NAG), a lysosomal enzyme of renal tubular origin, is a sensitive indicator of renal tubular function. A circadian rhythm in the urinary activity of NAG, statistically validated and quantified by the cosinor method, was documented in 19 female and 15 male human subjects. The acrophase of the circadian rhythm in urinary NAG activity occurs at 0940 with 95% confidence limits between 0840 and 1208 and is similar to the timing of the circadian rhythm in urinary free cortisol. The circadian acrophase of urinary NAG activity lags in timing the circadian rhythms in urine volume, Na and K excretion, and urinary free adrenalin [**epinephrine**] and noradrenalin [norepinephrine], by .apprx. 5-10 h and the circadian rhythm in creatinine excretion by .apprx. 11 h. These functions with their characteristic phase relations are part of the internal circadian time structure of the human organism and may provide internal phase references, independent of the time of day. This study documents a sex difference in mesor of the circadian rhythms in urinary NAG activity, with female subjects having a higher mesor and amplitude than the male subjects, and in the excretion of creatinine and K, with male subjects have a higher mesor and amplitude than the female subjects.

L5 ANSWER 72 OF 106 MEDLINE on STN
ACCESSION NUMBER: 83140977 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6298565
TITLE: Cholinergic, adrenergic, and PGE1 effects on cyclic nucleotides and growth in cultured corneal epithelium.
AUTHOR: Cavanagh H D; Colley A M
CONTRACT NUMBER: EY-01920 (NEI)
SOURCE: Metabolic, pediatric, and systemic ophthalmology, (1982) Vol. 6, No. 2, pp. 63-74.
Journal code: 8214904. ISSN: 0277-9382.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198304
ENTRY DATE: Entered STN: 18 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 7 Apr 1983

AB Effects of adrenergic and cholinergic drugs and prostaglandin E1 on cyclic nucleotide accumulation and parameters of growth and basement membrane synthesis were examined in corneal epithelial cell cultures. 8-bromo-cGMP significantly (p less than 0.05) enhanced incorporation of labeled thymidine and leucine, as did acetylcholine and carbamylcholine, which elevated cGMP and decreased cAMP/cGMP ratio. Responses to acetylcholine were abolished by atropine and alpha-bungarotoxin. Precursor incorporation was inhibited by dibutyryl cAMP and adenosine 5'-monophosphate and by norepinephrine, **epinephrine**, prostaglandin E1, and theophylline, which significantly elevated cAMP levels and cAMP/cGMP ratio. Propranolol, but not phenoxybenzamine, blocked responses to effective concentrations of norepinephrine. Norepinephrine, PGE1, and dibutyryl cAMP also significantly elevated uptake of labeled **glucosamine** and incorporation of labeled proline into collagenase-sensitive protein or the hydroxyproline fraction of protein hydrolysates, while acetylcholine had no effect on parameters of basement membrane synthesis. Propranolol blocked responses to norepinephrine. Results were consistent with a cGMP-mediated stimulatory role of the cholinergic transmitter in corneal epithelial growth regulation, cAMP-mediated beta-adrenergic suppression of regrowth and increased basement membrane production after initial injury to the corneal epithelium, and potentiation of the adrenergic effect by prostaglandins.

L5 ANSWER 73 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1981:114824 BIOSIS
DOCUMENT NUMBER: PREV198121049820; BR21:49820
TITLE: PLATELET SECRETION DEFECTS IN PATIENTS WITH MINIMAL BRAIN DYS FUNCTION.
AUTHOR(S): RAO A K [Reprint author]; KOIKE K; HOLMSEN H; MUELLER P
CORPORATE SOURCE: THROMBOSIS RESEARCH CENTER, TEMPLE UNIVERSITY HOSPITAL, PHILADELPHIA, PENNSYLVANIA, USA
SOURCE: Clinical Research, (1981) Vol. 29, No. 2, pp. 344A.
Meeting Info.: 38TH ANNUAL NATIONAL MEETING OF THE AMERICAN FEDERATION FOR CLINICAL RESEARCH, SAN FRANCISCO, CALIF., USA, APRIL 25-27, 1981. CLIN RES.
CODEN: CLREAS. ISSN: 0009-9279.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH

L5 ANSWER 74 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 20
ACCESSION NUMBER: 1981:581685 CAPLUS

DOCUMENT NUMBER: 95:181685
TITLE: The effects of **glucosamine** on platelet aggregation
AUTHOR(S): Bertram, J.; Ragatz, B. H.; Baldwin, W.; Iatrides, P. G.
CORPORATE SOURCE: Sch. Med., Indiana Univ., Gary, IN, 46408, USA
SOURCE: Thrombosis Research (1981), 23(3), 301-7
CODEN: THBRAA; ISSN: 0049-3848
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of **glucosamine**-HCl [66-84-2] on human platelet aggregation induced by a variety of agents were investigated. **Glucosamine** inhibited Staphylococcus aureus-induced platelet aggregation and release. **Glucosamine** also inhibited collagen, **epinephrine** [51-43-4], and ADP [58-64-0]-induced platelet aggregation and release.

L5 ANSWER 75 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 21
ACCESSION NUMBER: 1981:581684 CAPLUS
DOCUMENT NUMBER: 95:181684
TITLE: Effects of N-acetyl **glucosamine** on platelet aggregation
AUTHOR(S): Bertram, J.; Ragatz, B. H.; Baldwin, W.; Iatrides, P. G.
CORPORATE SOURCE: Sch. Med., Indiana Univ., Gary, IN, 46408, USA
SOURCE: Thrombosis Research (1981), 23(3), 289-300
CODEN: THBRAA; ISSN: 0049-3848
DOCUMENT TYPE: Journal
LANGUAGE: English
AB N-Acetylglucosamine (NAG) [7512-17-6] alone did not aggregate human platelets but in low concns. shortened the delay phase of the Staphylococcus aureus-induced platelet aggregation in a concentration-dependent manner, which suggests that NAG may be one of the components of the S. aureus cell wall responsible for platelet aggregation. NAG, on the other hand, inhibited collagen-induced platelet aggregation and the secondary platelet aggregation induced by **epinephrine** [51-43-4] and ADP [58-64-0]. This inhibition appears to be due to impurities that occur at higher levels in some NAG lots than in others. NAG also decreased the length of time before the release of ATP [56-65-5] from platelets when S. aureus was the aggregating agent.

L5 ANSWER 76 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 22
ACCESSION NUMBER: 1981:58591 CAPLUS
DOCUMENT NUMBER: 94:58591
TITLE: Immunologic and neuropharmacologic stimulation of mucous glycoprotein release from human airways in vitro
AUTHOR(S): Shelhamer, James H.; Marom, Zvi; Kaliner, Michael
CORPORATE SOURCE: Natl. Inst. Allergy Infect. Dis., NIH, Bethesda, MD, 20205, USA
SOURCE: Journal of Clinical Investigation (1980), 66(6), 1400-8
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English
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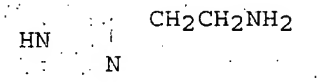


I

AB Human bronchial airways obtained after surgical resection were maintained in tissue culture for 24-48 h. Incorporation of [3H]- or [14C] **glucosamine**, [14C]threonine, or Na2[35S]O4 to the culture media resulted in biosynthesis of 2 radiolabeled glycoproteins: 1 filtering in the exclusion volume of Sepharose 2B, and the other filtering with an approx. mol. weight of 400,000. Both fractions had similar elution patterns from DEAE-cellulose anion exchange chromatog. [3H]**glucosamine** was incorporated equally into the 2 fractions. The effects of anaphylaxis, histamine diphosphate (I diphosphate) [51-74-1] and several neurohormones on the release of [3H]**glucosamine**-labeled glycoproteins were analyzed, making no attempt to sep. the 2 glycoprotein fractions. Three lines of evidence were found suggesting that mast cell degranulation increases mucous release from cultured airways. Supernatant fluids from anaphylaxed peripheral human lung that contained 200-400 ng I/mL and 400-1000 units slow-reacting substance of anaphylaxis (SRS-A)/mL increased the labeled glycoprotein release by 40%. The addition of antigen to IgE-sensitized airways led to the release of 26% of the total I and a 36% increase in mucous release. Reversed anaphylaxis with anti-IgE antibodies induced a 36% release of I from the airways and an increase in the release of mucous glycoproteins of 25%. Exogenous I added to airways increased mucous glycoprotein release, an effect prevented by cimetidine, an H2 antagonist. Selective I H2, but not H1 agonists increased mucous glycoprotein release, suggesting the possibility that anaphylaxis of airways results in increased mucous glycoprotein release partly through I H2 stimulation. A cholinomimetic, methacholine [55-92-5], increased mucous release; this response was prevented by atropine which alone had no effect. No response to β-adrenergic stimulation with either isoproterenol or **epinephrine** was noted. However, α-adrenergic stimulation with either norepinephrine [51-41-2] combined with propranolol or phenylephrine [59-42-7] alone resulted in dose-related increases in glycoprotein release. Both α-adrenergic and cholinergic stimulation of human tissues induce the formation of cyclic GMP [7665-99-8], and 8-bromo cyclic GMP [31356-94-2] added to the airways led to increased mucous secretion. Thus, it seems likely that neurohormones capable of stimulating cyclic GMP formation in human airways may lead to increased mucous glycoprotein release.

L5 ANSWER 77 OF 106 MEDLINE on STN DUPLICATE 23
ACCESSION NUMBER: 81059616 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6449150
TITLE: Acquired storage pool deficiency with increased platelet-associated IgG. Report of five cases.
AUTHOR: Weiss H J; Rosove M H; Lages B A; Kaplan K L
SOURCE: The American journal of medicine, (1980 Nov) Vol. 69, No. 5, pp. 711-7.
Journal code: 0267200. ISSN: 0002-9343.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; AIDS
ENTRY MONTH: 198101
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 16 Mar 1990
Entered Medline: 29 Jan 1981

AB Acquired abnormalities of platelet aggregation have been reported with increasing frequency. We studied five patients (including two with systemic lupus erythematosus and one with compensated chronic idiopathic thrombocytopenic purpura) in whom platelet aggregation responses to collagen, **epinephrine** and ADP are impaired; in all cases, we found that levels of platelet-associated immunoglobulin G (IgG) were

increased. In all five patients substances stored in platelet-dense granules (ATP, ADP, serotonin and calcium) were diminished. The content of the alpha-granule substance, beta-thromboglobulin, was also decreased in most cases, whereas the levels of two secretable acid hydrolase enzymes (beta-glucuronidase and beta-N-acetyl **glucosaminidase**) were within normal limits. These findings are similar to those observed in subtypes of congenital storage pool deficiency. However, in contrast to the congenital disorder, a membrane-bound (nonsecretable) acid phosphatase was also decreased in the patients with acquired storage pool deficiency. These findings suggest that impaired platelet aggregation on an acquired basis may, in some patients, be due to immune platelet damage resulting in a distinctive type of platelet storage pool deficiency.

L5 ANSWER 78 OF 106 MEDLINE on STN DUPLICATE 24
ACCESSION NUMBER: 80172809 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6102846
TITLE: Secretory response of dispersed rat submandibular cells. II. Mucin secretion.
AUTHOR: Quissell D O; Barzen K A
SOURCE: The American journal of physiology, (1980 Mar) Vol. 238, No. 3, pp. C99-106.
Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198006
ENTRY DATE: Entered STN: 15 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 27 Jun 1980

AB The secretory response of dispersed rat submandibular cells as it relates to the secretion of D-[1-14C]**glucosamine** hydrochloride-labeled mucin following sympathomimetic and parasympathomimetic stimulation was evaluated. The adrenergic agonists (-)-norepinephrine and (-)-**epinephrine** were found to have equal efficacy and potency with a median effective concentration (EC50) of 7.1 x 10(-7) M. (-)-Isoproterenol was found to be acting as a "partial" agonist and had an EC50 of 3.9 x 10(-7) M. (-)-Phenylephrine addition resulted in a small, but significant, secretion of mucin at higher doses tested (10(-4) M--10(-3) M). Neither cholinergic nor alpha-adrenergic receptor stimulation was able to elicit a net increase in the secretion of mucin. However alpha-adrenergic receptor activation in conjunction with beta-adrenergic receptor activation facilitated the rate of secretion. Extracellular Ca2+ and Mg2+ were not required for the secretion of mucin, but extracellular Ca2+ enhanced the rate of secretion following alpha- and beta-adrenergic receptor activation. However extracellular Ca2+ did not enhance mucin secretion following beta-adrenergic receptor activation. Both cellular Ca2+ and beta-adrenergic receptor activation were required to elicit a secretory response following sympathomimetic stimulation.

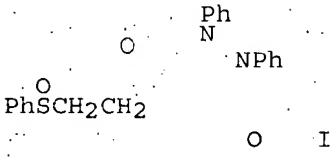
L5 ANSWER 79 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 25
ACCESSION NUMBER: 1980:1954 CAPLUS
DOCUMENT NUMBER: 92:1954
TITLE: Purification and characterization of a lectin from rice bran
AUTHOR(S): Tsuda, Masao
CORPORATE SOURCE: Biol. Res. Lab., Takeda Chem. Ind., Ltd., Osaka, 532, Japan
SOURCE: Journal of Biochemistry (Tokyo, Japan) (1979), 86(5), 1451-61

CODEN: JOBIAO; ISSN: 0021-924X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A rice bran lectin was purified to homogeneity by precipitation with (NH4)2SO4 and chromatog. on ovomucoid-Sepharose and CM-cellulose. The mol. weight of the dimer lectin was estimated to be .apprx.37,000 by ultracentrifugation studies. The sedimentation coefficient was 3.8 S. On sepharose 6B gel filtration in the presence of 6M guanidine-HCl, the lectin showed a mol. weight of 19,000. On reduction and carboxymethylation, the lectin further dissociated into 2 nonidentical subunits, with mol. wts. of .apprx.11,000 and 8000. These subunits did not show hemagglutinating activity. Equilibrium dialysis expts. using N-acetyl-**glucosamine**-1-14C indicated that .apprx.1.8 mol of the sugar was bound to 19,000 g of the lectin. The lectin was mitogenic against mouse splenic lymphocytes and human peripheral lymphocytes. The lectin enhanced the rate of glucose oxidation and inhibited **epinephrine**-stimulated lipolysis in mouse adipocytes. Some characteristics of the lectin are compared with those of wheat germ agglutinin.

L5 ANSWER 80 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 26
ACCESSION NUMBER: 1979:604875 CAPLUS
DOCUMENT NUMBER: 91:204875
TITLE: In vitro response of enzyme-dispersed rat intestinal villus and crypt cells to hormones
AUTHOR(S): Hoffman, Alan G. D.; Kuksis, Arnis
CORPORATE SOURCE: Charles H. Best Inst., Univ. Toronto, Toronto, ON, M5G 1L6, Can.
SOURCE: Canadian Journal of Physiology and Pharmacology (1979), 57(12), 1393-400
CODEN: CJPPA3; ISSN: 0008-4212
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Villus and crypt cells isolated from rat jetjunal tissue by an improved enzymic procedure were tested for their ability to respond to hormonal stimulation using several metabolic tracers. Incubation with cyclic AMP [60-92-4], theophylline [58-55-9], isoproterenol [7683-59-2], and **epinephrine** bitartrate [51-42-3] resulted in 20-70% increases in incorporation of D-**glucosamine** into glycoproteins. The newly formed material was largely (90-95%) recovered from the medium, apparently as a result of active secretion, because tests for cellular leakage and breakage failed to account for it. The above hormones and pharmacol. agents as well as glucagon [9007-92-5], insulin [9004-10-8], and cortisol [50-23-7], however, failed to modify the rate of incorporation of acetate and glucose into the total cellular lipids of the isolated crypt and villus cells. Likewise, segments of intestinal tissue, although readily responding with increased incorporation of D-**glucosamine** (50-100%) failed to demonstrate any effect of added hormones on the incorporation of acetate even when lipogenesis was stimulated by addition of glucose or fructose. Apparently, these enzyme-dispersed cells are suitable for metabolic studies.

L5 ANSWER 81 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1979:180017 CAPLUS
DOCUMENT NUMBER: 90:180017
TITLE: The effect of sulfinpyrazone on the aggregation and release reactions of human platelets
AUTHOR(S): Wiley, J. S.; Chesterman, C. N.; Morgan, F. J.; Castaldi, P. A.
CORPORATE SOURCE: Dep. Med., Univ. Melbourne, Heidelberg, Australia
SOURCE: Thrombosis Research (1979), 14(1), 23-33
CODEN: THBRAA; ISSN: 0049-3848

DOCUMENT TYPE: Journal
LANGUAGE: English
GI

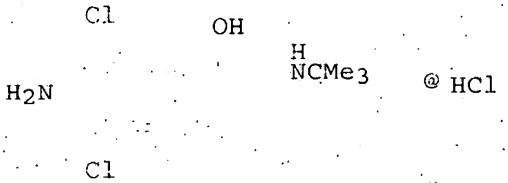


AB Sulfinpyrazone (I) [57-96-5] at low concns. (1-5 μM) inhibited the in vitro aggregation of normal platelets induced by **epinephrine**, collagen, and ristocetin. At higher concns. (>5 μM) I also inhibited the thrombin-induced release of blood platelet factor 4 [37270-94-3], β-thromboglobulin, serotonin [50-67-9], and β-N-acetyl **glucosaminidase** [9012-33-3] from an unstirred suspension of gel-filtered platelets. The inhibition of release or aggregation could be overcome by increasing the concentration of aggregating agent. I had no effect on the active or passive influx of K+ into the platelet. These inhibitory effects were observed at concns. of I likely to be reached by the daily administration of 800 mg.

L5 ANSWER 82 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1980:160135 BIOSIS
DOCUMENT NUMBER: PREV198069035131; BA69:35131
TITLE: EFFECT OF CATECHOLAMINES ON THE ACTIVITY OF LYSOSOMAL ENZYMES IN THE BLOOD SERUM OF RATS CHRONICALLY TREATED WITH HYDRAZINO PHTHALAZINES.
AUTHOR(S): DROZDZ M [Reprint author]; KUCHARZ E; OLCZYK K
CORPORATE SOURCE: DEP CLIN CHEM, INST PHARMACODYN MED ANAL, SILESIAN MED ACAD, JAGIELLONSKA 4, 41-200 SOSNOWIEC, POL
SOURCE: Folia Biologica (Cracow), (1979) Vol. 27, No. 1, pp. 9-16. CODEN: FOBGA8. ISSN: 0015-5497.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB The effect of a single injection of noradrenaline [norepinephrine] or adrenaline [**epinephrine**] on the activity of lysosomal enzymes (β-glucuronidase, β-acetylglucosaminidase, hyaluronidase and arylsulfatase) was studied in the blood serum of nomral and experimental rats chronically treated with the collagen-like syndrome inductors hydralazine (1-hydrazinophthalazine) or binazine (N1-ethoxycarbonyl-N2-hydrazinophthalazine). Administration of catecholamines to healthy rats stimulated increase in the activity of all the studied enzymes, the greatest changes being shown after adrenaline injection. Long-term treatment with hydralazine produced changes in the activity of lysosomal enzymes, while administration of catecholamines decreased this activity. Smaller changes in enzyme activity found after binazine treatment were probably due to the low toxicity of this compound with respect to connective tissue. The mechanism of the observed disturbances is discussed.

L5 ANSWER 83 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 27
ACCESSION NUMBER: 1979:97453 CAPLUS
DOCUMENT NUMBER: 90:97453
TITLE: Histochemical experiments on the action of **clenbuterol** on tracheal mucosa
AUTHOR(S): Schaetzle, W.; Federspil, P.

CORPORATE SOURCE: Hals-Nasen-Ohren-Klin., Univ. Homburg, Homburg/Saar, Fed. Rep. Ger.
SOURCE: Arzneimittel-Forschung (1978), 28(7), 1116-19
CODEN: ARZNAD; ISSN: 0004-4172
DOCUMENT TYPE: Journal
LANGUAGE: German
GI



I

AB **Clenbuterol**-HCl (I) [21898-19-1] (4 mg/day for 5 days or 1 mg/day for 12 or 28 days) injected i.p. into guinea pigs increased total acid phosphatase [9001-77-8] and N-acetyl-β-D-**glucosaminidase** [9012-33-3] in mucosa of trachea epithelium. I had a min. to moderate effect on nonspecific esterase [9013-79-0] activity and alkaline phosphatase [9001-78-9] was increased only in the long-term test, mainly in capillaries. The long-term test also increased RNA in tracheal mucosal epithelium. I decreased the number of stainable goblet cells of the mucosa and part of the acid mucous components containing neuraminic acid rose of the acid mucous components containing neuraminic acid rose so much that a qual. change in the direction of neuraminidase-sensitive acid glycosaminoglycans was demonstrated.

L5 ANSWER 84 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1979:20127 CAPLUS
DOCUMENT NUMBER: 90:20127
TITLE: Effect of β-hydroxybutyric acid on lipolysis and glucose metabolism in incubated fat tissue from rats
AUTHOR(S): Spiroski, M. Z.; Kovacev, V. P.; Bitoljanu, P.
CORPORATE SOURCE: Med. Fak., Univ. Cent. Med. Nauka, Skopje, Yugoslavia
SOURCE: Diabetol. Croat., Supl. (1977), 1(Zb. Rad. Jugosl. Simp. Secernoj Boles., 3rd), 381-4
CODEN: DCSUDW
DOCUMENT TYPE: Journal
LANGUAGE: Serbo-Croatian
AB In isolated rat adipose tissue, β-hydroxybutyrate (0.5 mM) inhibited **epinephrine**-induced free fatty acid release, glycerol release, and glucose incorporation into CO2 and neutral lipids. Ketone bodies may have an autoregulatory role in their own production

L5 ANSWER 85 OF 106 MEDLINE on STN
ACCESSION NUMBER: 78082290 MEDLINE
DOCUMENT NUMBER: PubMed ID: 413629
TITLE: Neutral and acid lipase of mouse 3T3 fibroblasts with increased adipose conversion.
AUTHOR: Adebonojo F O; Cortner J A; Coates P M
SOURCE: Cell differentiation, (1977 Dec) Vol. 6, No. 5-6, pp. 307-12.
Journal code: 0342640. ISSN: 0045-6039.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 197803
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 21 Mar 1978

AB In the resting state, 3T3-L1 fibroblasts become adipose converted and increase their fatty acid and triglyceride synthetase. We have found that they contain four times the neutral lipase activity and 1.5 times the acid lipase activity of logarithmically dividing cells. The activities of lysosomal acid beta-galactosidase and N-acetyl-beta-D-**glucosaminidase** were the same in the adipose converted and logarithmically dividing cells. The data suggest a possible relation between the increased neutral lipase activity in 3T3-L1 cells and their adipose conversion and demonstrates that the adipose converted 3T3-L1 fibroblasts, unlike true adipose cells, contain high levels of lysosomal acid hydrolases.

L5 ANSWER 86 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 28
ACCESSION NUMBER: 1976:504648 CAPLUS
DOCUMENT NUMBER: 85:104648
TITLE: Studies on the inhibition of glycogenolysis by D-galactosamine in rat liver hepatocytes
AUTHOR(S): Wagle, S. R.; Stermann, R.; Decker, K.
CORPORATE SOURCE: Med. Fak., Univ. Freiburg, Freiburg/Br., Fed. Rep. Ger.
SOURCE: Biochemical and Biophysical Research Communications (1976), 71(2), 622-8
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB At 1 mM, D-galactosamine [7535-00-4] almost completely inhibited glucose [50-99-7] formation in isolated rat hepatocytes; this inhibition was not removed by addition of glucagon, **epinephrine**, cyclic AMP, cyclic GMP, or cyclic UMP to the medium. D-galactose, 2-deoxyglucose, and D-**glucosamine** had no effect on glycogenolysis in the isolated hepatocytes. The inhibitory effect of D-galactosamine was due neither to an impaired cyclic AMP production nor to an alteration of the metabolism of glucose-1-phosphate.

L5 ANSWER 87 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1976:493285 CAPLUS
DOCUMENT NUMBER: 85:93285
TITLE: Detection of molecular ions of thermally unstable compounds by in beam electron impact
AUTHOR(S): Ohashi, Mamoru; Tsujimoto, Kazuo; Yasuda, Akiyoshi
CORPORATE SOURCE: Dep. Mater. Sci., Univ. Electro-Commun., Chofu, Japan
SOURCE: Chemistry Letters (1976), (5), 439-40
CODEN: CMLTAG; ISSN: 0366-7022
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The in-beam electron impact spectra of adenosine, **ephedrine**-HCl, L-fucose, **glucosaminic** acid, L-glutamic acid, guanosine, inosine, N-methylephedrine, quinine, sucrose, and D-xylose were determined The spectra almost always exhibited the M+1 peak in place of M. Intensities of M+1 peaks of several thermally unstable compds., for which mol. ion peaks were not observed with conventional direct inlet systems, are reported.

L5 ANSWER 88 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 29
ACCESSION NUMBER: 1974:433934 CAPLUS
DOCUMENT NUMBER: 81:33934
TITLE: Pancreatic β-cell recognition of insulin secretagogues. VIII. Comparisons of glucose with glyceraldehyde isomers and dihydroxyacetone

AUTHOR(S): Hellman, Bo; Idahl, Lars A.; Lernmark, Ake; Sehlin, Janove; Taljedal, Inge B.
CORPORATE SOURCE: Dep. Histol., Univ. Umed, Umed, Swed.
SOURCE: Archives of Biochemistry and Biophysics (1974), 162(2), 448-57
CODEN: ABBIA4; ISSN: 0003-9861
DOCUMENT TYPE: Journal
LANGUAGE: English

AB D-glyceraldehyde [453-17-8] stimulated the release of insulin [9004-10-8] from isolated mouse pancreatic islets whether or not glucose [50-99-7] was present in the medium. Like the action of glucose, that of D-glyceraldehyde was biphasic in time, exhibited a sigmoidal dose-response relation, was potentiated by theophylline, arginine, iodoacetamide, or L-glyceraldehyde, and was inhibited by **epinephrine**, 2,4-dinitrophenol, or Ca2+ deficiency. Half-maximum and maximum stimulations were produced by about 3 mM and 10 mM D-glyceraldehyde. Pos. interactions were observed between 5 mM D-glyceraldehyde and 5mM glucose and between 10 mM D-glyceraldehyde and 10 mM leucine. Mannoheptulose (10 mM) or **glucosamine** (10 mM) potentiated the effect of 10 mM D-glyceraldehyde. 1,3-Dihydroxyacetone [96-26-4] (2.5-20 mM) also initiated insulin release in the absence of glucose; 5-10 mM L-glyceraldehyde [497-09-6] did not initiate secretion but potentiated the effects of 5 mM glucose or 5 mM D-glyceraldehyde. D-glyceraldehyde or dihydroxyacetone reduced the production of 14CO2 from D-[U-14C]glucose. The results suggest that by being phosphorylated and entering glycolysis in the β-cells, D-glyceraldehyde and dihydroxyacetone act as functional analogs of glucose in secretory stimulation. Initiation of insulin release by glucose, D-glyceraldehyde, or dihydroxyacetone may thus depend on the production of a metabolic signal at or below the triose phosphate level.

L5 ANSWER 89 OF 106 MEDLINE on STN
ACCESSION NUMBER: 73096302 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4510292
TITLE: Insulin-like activity of concanavalin A and wheat germ agglutinin--direct interactions with insulin receptors.
AUTHOR: Cuatrecasas P; Tell G P
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1973 Feb) Vol. 70, No. 2, pp. 485-9.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197304
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 5 Apr 1973

AB Concanavalin A and wheat germ agglutinin are as effective as insulin in enhancing the rate of glucose transport and in inhibiting **epinephrine**-stimulated lipolysis in isolated adipocytes. These lectins, also like insulin, inhibit basal as well as **epinephrine**-stimulated adenylate cyclase activity of membranes obtained from homogenates of fat cells. Low concentrations of wheat germ agglutinin enhance the specific binding of insulin to receptors of fat cells and liver membranes. Higher concentrations of this plant lectin, as well as of concanavalin A, competitively displace the binding of insulin to receptors in these tissues. These effects are equally apparent in insulin-binding proteins solubilized from membranes, indicating that the plant lectins interact directly with insulin receptors. All of the effects observed with the plant lectins are reversed by simple sugars that

bind specifically to these plant proteins. Agarose derivatives of the plant lectins effectively adsorb solubilized insulin-binding proteins, and these can be eluted with buffers containing specific simple sugars. The possible implications of these findings to certain biological properties (mitogenicity) of these lectins and to the mechanism of action of other growth-promoting substances are considered.

L5 ANSWER 90 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 30
ACCESSION NUMBER: 1973:154728 CAPLUS
DOCUMENT NUMBER: 78:154728
TITLE: Cyclic AMP and intestinal glycoprotein synthesis. Effect of β-adrenergic agents, theophylline, and dibutyryl cyclic AMP
AUTHOR(S): Forstner, Gordon; Shih, Michael; Lukie, Bryan
CORPORATE SOURCE: Gastrointest. Unit, Toronto West. Hosp., Toronto, ON, Can.
SOURCE: Canadian Journal of Physiology and Pharmacology (1973), 51(2), 122-9
CODEN: CJPPA3; ISSN: 0008-4212
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Isoproterenol (I) [7683-59-2] (10-4M), **epinephrine** [51-43-4] (10-4M), theophylline [58-55-9] (10-3M), and dibutyryl cyclic AMP [362-74-3] (10-3M) increased the incorporation of 14C-labeled **glucosamine** [3416-24-8] into acid precipitable protein by rat intestinal slices. The effect of either I or **epinephrine** was potentiated by the addition of theophylline. The uptake of **glucosamine** into the acid soluble compartment of intestinal slices was not affected by I, **epinephrine**, or theophylline. Cycloheximide [66-81-9] (10-2M) inhibited the stimulatory effect of I and theophylline. Subfractionation of intestinal slice homogenates indicated that increased synthesis was not confined to one glycoprotein class but appeared to affect mucins and glycocalyx glycoproteins equally. β-Adrenergic hormones and cyclic AMP [60-92-4] stimulate intestinal glycoprotein synthesis and may control the production of glycoproteins located at the intestinal surface.

L5 ANSWER 91 OF 106 MEDLINE on STN
ACCESSION NUMBER: 75123561 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4805214
TITLE: [Regulation of AMP-aminohydrolase activity in soluble brain fractions].
Reguliatsiia AMF-aminogidrolaznoi aktivnosti v rastvorimoi fraktsii mozga.
AUTHOR: Arutiunian A V; Gulian E A; Manukian L A; Nersisian Ts M
SOURCE: Voprosy biokhimii mozga, (1973) Vol. 8, pp. 63-76.
Journal code: 0036162. ISSN: 0507-2972.
PUB. COUNTRY: USSR
DOCUMENT TYPE: (COMPARATIVE STUDY)
(ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197505
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 23 May 1975

L5 ANSWER 92 OF 106 MEDLINE on STN
ACCESSION NUMBER: 74109152 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4677554
TITLE: Ultrastructural changes in organ cultures of bovine veins.

AUTHOR: Zwillenberg H H; Zwillenberg L O; Laszt L
SOURCE: Angiologica, (1972) Vol. 9, No. 3-6, pp. 292-300.
Journal code: 0427125. ISSN: 0003-3189.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197404
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 29 Apr 1974

L5 ANSWER 93 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1971:97529 CAPLUS
DOCUMENT NUMBER: 74:97529
TITLE: Metabolic alterations accompanying α-amylase secretion by rat parotid tissue in vitro
AUTHOR(S): Ueha, Takao; Catanzaro, Orlando; Hanson, Roger; Lindsay, Raymond H.
CORPORATE SOURCE: Dep. Pharmacol. Med., Univ. Alabama, Birmingham, AL, USA
SOURCE: American Journal of Physiology (1971), 220(2), 312-18
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
GI For diagram(s), see printed CA Issue.
AB The addition of pilocarpine (I) (10 μg/ml) and **epinephrine** (II) (3.3 μg/ml) to rat parotid gland prepns. in vitro resulted in a loss of α-amylase from the tissue and an increase in enzyme in the incubation medium. I and II, depending on the concentration, increased oxidation of glucose ≤160, of various amino acids ≤100%, and of β-hydroxybutyrate ≤40%. However, incorporation of 14C-labeled amino acid or **glucosamine** into tissue protein was depressed 20-40%. Conversion of carboxyl-14C-labeled orotic acid to pyrimidine nucleotides was also depressed with higher concns. of these drugs. Apparently, stimulation of parotid α-amylase secretion in vitro is accompanied not only by an increase in the oxidation of glucose and amino acids but also by a decrease in the synthesis of glycoprotein and pyrimidine nucleotides.

L5 ANSWER 94 OF 106 MEDLINE on STN
ACCESSION NUMBER: 71010943 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4919469
TITLE: Glucose metabolism in mouse pancreatic islets.
AUTHOR: Ashcroft S J; Hedeskov C J; Randle P J
SOURCE: The Biochemical journal, (1970 Jun) Vol. 118, No. 1, pp. 143-54.
Journal code: 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197012
ENTRY DATE: Entered STN: 1 Jan 1990
Last Updated on STN: 1 Jan 1990
Entered Medline: 8 Dec 1970
AB 1. Rates of glucose oxidation, lactate output and the intracellular concentration of glucose 6-phosphate were measured in mouse pancreatic islets incubated in vitro. 2. Glucose oxidation rate, measured as the formation of (14)CO(2) from [U-(14)C]glucose, was markedly dependent on

extracellular glucose concentration. It was especially sensitive to glucose concentrations between 1 and 2mg/ml. Glucose oxidation was inhibited by mannoheptulose and **glucosamine** but not by phlorrhizin, 2-deoxyglucose or N-acetylglucosamine. Glucose oxidation was slightly stimulated by tolbutamide but was not significantly affected by adrenaline, diazoxide or absence of Ca(2+) (all of which may inhibit glucose-stimulated insulin release), by arginine or glucagon (which may stimulate insulin release) or by cycloheximide (which may inhibit insulin synthesis). 3. Rates of lactate formation were dependent on the extracellular glucose concentration and were decreased by **glucosamine** though not by mannoheptulose; tolbutamide increased the rate of lactate output. 4. Islet glucose 6-phosphate concentration was also markedly dependent on extracellular glucose concentration and was diminished by mannoheptulose or **glucosamine**; tolbutamide and glucagon were without significant effect. Mannose increased islet fructose 6-phosphate concentration but had little effect on islet glucose 6-phosphate concentration. Fructose increased islet glucose 6-phosphate concentration but to a much smaller extent than did glucose. 5. [1-(14)C]Mannose and [U-(14)C]fructose were also oxidized by islets but less rapidly than glucose. Conversion of [1-(14)C]mannose into [1-(14)C]glucose 6-phosphate or [1-(14)C]glucose could not be detected. It is concluded that metabolism of mannose is associated with poor equilibration between fructose 6-phosphate and glucose 6-phosphate. 6. These results are consistent with the idea that glucose utilization in mouse islets may be limited by the rate of glucose phosphorylation, that mannoheptulose and **glucosamine** may inhibit glucose phosphorylation and that effects of glucose on insulin release may be mediated through metabolism of the sugar.

L5 ANSWER 95 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1971:446717 CAPLUS
DOCUMENT NUMBER: 75:46717
TITLE: Mucopolysaccharides (glycosaminoglycans) of the cornea in some pathological states
AUTHOR(S): Kasavina, B. S.; Vinetskaya, M. I.
CORPORATE SOURCE: Nauchno-Issled. Inst. Glaznykh Bolezn. im. Gel'mgol'tsa, Moscow, USSR
SOURCE: Uchenye Trudy Gor'kovskogo Gosudarstvennogo Meditsinskogo Instituta im. S. M. Kirova (1970), No. 32, 42-6
CODEN: UTGMBE; ISSN: 0434-2038
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The total amount of hexosamines and uronic acids, as the indicators of chondroitin sulfate and chondroitin of mucopolysaccharides (MP), was studied. The qual. distribution of glucose- and galactose-containing MP was investigated by chromatog. on ion exchangers. After trigeminectomy a significant decrease in MP was observed (62% of controls), together with a decrease in keratosulfate and an increase in chondroitin sulfate and chondroitin concns. During the formation of the scar the ratio **glucosamine**/galactosamine in the cornea was changed from 2.04 to 0.82. Some tendency towards normalization was observed when ascorbic acid was given to the exptl. animals. The total MP content was decreased when glucoroticoids were applied, and also after i.v. injection of **epinephrine.**

L5 ANSWER 96 OF 106 MEDLINE on STN
ACCESSION NUMBER: 71080102 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4923965
TITLE: Factors controlling insulin secretion in vitro.
AUTHOR: Martin J M
SOURCE: Acta diabetologica latina, (1969 Oct-Dec) Vol. 6, No. 4,

pp. 689-712.
Journal code: 0123567. ISSN: 0001-5563.
PUB. COUNTRY: Italy
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: MULTILINGUAL
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197102
ENTRY DATE: Entered STN: 1 Jan 1990
Last Updated on STN: 1 Jan 1990
Entered Medline: 27 Feb 1971

L5 ANSWER 97 OF 106 MEDLINE on STN
ACCESSION NUMBER: 69262569 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4308407
TITLE: Studies on the ability of compounds to block the diabetogenic activity of streptozotocin.
AUTHOR: Dulin W E; Wyse B M
SOURCE: Diabetes, (1969 Jul) Vol. 18, No. 7, pp. 459-66.
Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 196909
ENTRY DATE: Entered STN: 1 Jan 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 30 Sep 1969

L5 ANSWER 98 OF 106 MEDLINE on STN
ACCESSION NUMBER: 70133927 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5212265
TITLE: Inhibitors of insulin secretion.
AUTHOR: Frerichs H; Creutzfeldt C; Creutzfeldt W
SOURCE: Acta diabetologica latina, (1968 Oct) Vol. 5 Suppl 1, pp. 105-23.
Journal code: 0123567. ISSN: 0001-5563.
PUB. COUNTRY: Italy
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: MULTILINGUAL
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197004
ENTRY DATE: Entered STN: 1 Jan 1990
Last Updated on STN: 1 Jan 1990
Entered Medline: 24 Apr 1970

L5 ANSWER 99 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 31
ACCESSION NUMBER: 1965:501193 CAPLUS
DOCUMENT NUMBER: 63:101193
ORIGINAL REFERENCE NO.: 63:18679d-f
TITLE: Polysaccharide-lipid complexes from Veillonella parvula
AUTHOR(S): Mergenhagen, Stephan E.
CORPORATE SOURCE: U.S. Public Health Serv., Bethesda, MD
SOURCE: Journal of Bacteriology (1965), 90(6), 1730-4
CODEN: JOBAAY; ISSN: 0021-9193
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A strain of V. parvula elaborates an extracellular slime when grown in a nutrient medium containing only dialyzable components. Deproteinization with CHCl3-BuOH of EtOH-precipitated material from the supernatant culture fluid leads

to the isolation of a watersol. lipopolysaccharide (LPS1). Another component (LPS2), showing similarity in biol. and immunological properties to the endotoxic antigen (LPC) isolated from whole cells, was extracted with phenol from the insol. emulsion remaining after CHCl3BuOH extraction of slime. Analysis of polysaccharides by thinlayer chromatography demonstrated the presence of glucose and galactose in LPS1 and glucose, **glucosamine**, galactosamine, and a methyl pentose in LPC. LPS1 failed to give a pos. **epinephrine** skin test after intravenous injection in rabbits and failed to kill pertussis-sensitized mice, whereas LPS2 and LPC were active in both of these bioassays. Both lipopolysaccharides (LPS1 and LPC) exhibited type-specific haptenic activity in hemagglutination tests with numerous anti-Veillonella rabbit serums. LPS1 was found in these tests to be unrelated to a heterologous strain of Veillonella possessing a related somatic antigen. These expts. reveal the presence of two chemically and immunologically distinguishable polysaccharide-lipid complexes in this strain of V. parvula.

L5 ANSWER 100 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1964:94743 BIOSIS
DOCUMENT NUMBER: PREV19644500094750; BA45:94750
TITLE: Biochemistry of the alligator. A study of metabolism in slow motion.
AUTHOR(S): COULSON, ROLAND A.; HERNANDEZ, THOMAS
SOURCE: (1964) pp. xx+138p. Illus. Biochemistry of the alligator. A study of metabolism in slow motion.
Publisher: Louisiana State University Press, Baton Rouge 3, Louisiana.
DOCUMENT TYPE: Book
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB This work not only sheds light on a new animal species, biochemically speaking, but thereby sheds new light on old problems such as the comparability of in vitro and in vivo experiments. The latter type is facilitated in the alligator by its slow motion metabolism. This makes more evident the discrepancy between rapid metabolism of certain amino acids in tissue extracts and their slow turnover in the intact animal. The alligator's metabolism of glucose and other sugars was compared to that of the rat, with particular attention to the pronounced diabetogenic effect of **glucosamine.** Amino acid utilization proceeded at about one-fiftieth the rate seen in rats. Detailed studies of kidney function revealed the prominent role of NH_3 synthesis. Greater extremes of blood pH and plasma HCO_3 level were found in the alligator than in any other known species. Generalized metabolic effects of insulin and **epinephrine** became more discernible in these slow motion studies. The relation of the new findings to general biological theories is provided in discussion sections. ABSTRACT AUTHORS: J. R. Pleasants

L5 ANSWER 101 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1966:36755 BIOSIS
DOCUMENT NUMBER: PREV19664700036757; BA47:36757
TITLE: Regulation of insulin secretion studied with pieces of rabbit pancreas incubated in vitro.
AUTHOR(S): COORE, H. G.; RANDLE, P. J.
CORPORATE SOURCE: Dep. Physiol., Univ. West Indies, Mona, Jamaica
SOURCE: BIOCHEM J, (1964) Vol. 93, No. 1, pp. 66-78.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB The effect of various factors on the rate of release of insulin from pieces of rabbit pancreas incubated in vitro has been studied by estimating the insulin concentration in the medium after incubation by immunological assay with insulin-antibody precipitate, and the conditions necessary for eliciting consistent responses have been defined. Insulin release was accelerated by D-glucose at concentrations above 0.35-0.7 mg/ml and by D-mannose (3 or 6 mg/ml), but not by D-galac-tose, 3-O-methyo-D-glucose, D-fructose, D-ribose or sodium D-gluconate (3 mg/ml), D-2-deoxyglucose (3 or 6 mg/ml), N-acetyl-D-**glucosamine** (15 mg/ml) or D-mannoheptulose (3 mg/ml). The stimulating effect of glucose (3 mg/ml) on insulin release was abolished by mannoheptulose but not by 2-deoxyglucose, 3-O-methylglucose, ribose or N-acetylglucosamine. Insulin release at a low glucose concentration (0.6 mg/ml) was accelerated by tolbutamide (200 [mu]g/ml) but not by anoxia, 2,4-dinitrophenol, salicylate, p-phenylenediamine or phenazine methosulphate. The rate of insulin release at a high glucose concentration (3 mg/ml) was markedly diminished by anoxia, 2,4-dinitrophenol (250 [mu]M), salicylate (5 mM), p-phenylenediamine (1 mM) and phenazine methosulphate (100 [mu]M), but not by malonate (10 mM). The stimulating effect of tolbutamide (unlike that of glucose) was not influenced by mannoheptulose. The stimulating effect of glucose (3 mg/ml) on insulin release was augmented by the presence in the medium of glutamate, fumarate and pyruvate. At a low glucose concentration (0.6 mg/ml) neither these acids nor octanoate, acetoacetate or [beta] -hydroxybutyrate influenced the rate of insulin release. The stimulating effect of glucose (3 mg/ml) on insulin release was abolished by **epinephrine** (200 [mu]mg/ ml), and the effect of norepinephrine was suppressed by ergotamine tartrate (2.8 [mu]g/ml). Evidence is presented that the release of insulin by rabbit pancreas in vitro provides a suitable model for the behavior of [beta] -cells in vivo and for studying the influence of various factors on insulin secretion. The possible roles of glucose phosphorylation and of pathways of metabolism of glucose 6-phosphate in [beta] -cells in the stimulation of insulin release induced by glucose are discussed. ABSTRACT AUTHORS: Authors

L5 ANSWER 102 OF 106 MEDLINE on STN
ACCESSION NUMBER: 64031744 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14074107
TITLE: THE EFFECT OF HEPARIN AND ITS COMPONENTS ON FROG HEART.
AUTHOR: CSABA G; HORVATH C
SOURCE: Biochemical pharmacology, (1963 Oct) Vol. 12, pp. 1075-80. Journal code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: OLDMEDLINE; NONMEDLINE
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 16 Jul 1999
Last Updated on STN: 16 Jul 1999
Entered Medline: 1 Dec 1996

L5 ANSWER 103 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1963:28671 CAPLUS
DOCUMENT NUMBER: 58:28671
ORIGINAL REFERENCE NO.: 58:4840a-d
TITLE: Comparative biochemistry of parasitic helminths
AUTHOR(S): Bueding, Ernest
CORPORATE SOURCE: Johns Hopkins Univ., Baltimore, MD
SOURCE: Comparative Biochemistry and Physiology (1962), 4, 343-51

CODEN: CBCPAI; ISSN: 0010-406X
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Phosphoglucose isomerase of schistosomes and their mammalian host have identical kinetic properties but different immunological properties and active centers of the enzyme. Lactic dehydrogenases of schistosomes and their mammalian hosts have differences in kinetics, pH optima, substrate specificity, and cofactor requirements. There are 4 different hexokinases in schistosomes specific for glucose, fructose, mannose, **glucosamine**, while the mammalian and yeast enzymes catalyze the phosphorylation of a variety of hexoses. Schistosomes have a highly specific protease for hemoglobin or globin. Differential susceptibility to inhibitors is also a characteristic of the schistosomes and host enzymes. Antimonials inhibit the activity of phosphofructokinase of Schistosoma mansoni. Biochemical differences in Fasciola hepatica were observed. The major products of anaerobic glucose metabolism were propionic and acetic acids. Low concns. of 5-hydroxytryptamine produced and increased lactate production and the formation of cyclic adenosine 3,5-phosphate(3,5-AMP); this effect was not observed in mammalian enzymes. **Epinephrine** stimulated the accumulation of 3,5-AMP in mammalian enzymes, having no effect in parasite enzymes. 3,5-AMP had a stimulating effect on both parasites and host phosphorylase. The conversion of fats into carbohydrates was demonstrated, when Ascaris lumbricoides eggs develop to the infective stage. Although the habitat of Ascaris is anaerobic, the O uptake is ascribed to the oxidation of succinate present in the perienteric fluid by a succinic oxidase system where no cytochrome c or cytochrome oxidase could be detected. The production of H_2O_2, the increase in activity when incubation is done in pure O, and the lack of inhibition by CN^- or antimycin a suggests that the particulate succinic oxidase activity of A scaris is a flavoprotein. This enzyme is stimulated by Mn, inhibited by chelators, and the inhibition is reversed by Mn. There is a reduced diphosphopyridine nucleotide activity present, which also increases in activity by incubation in pure O and produces H_2O_2. The electron-transport properties of both enzymes are discussed, as well as some isolation properties.

L5 ANSWER 104 OF 106 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1962:54471 BIOSIS
DOCUMENT NUMBER: PREV19623900004739; BA39:4739
TITLE: Selective displacement of dye on biogenic polyelectrolytes as a model for the liberation of biogenic amines. Original Title: Selektive Farbstoffverdrangung an biogenen Polyelektroly-ten als Modell far die Liberation biogener Amine.
AUTHOR(S): JAQUES, R.; KUTTNER, K.
CORPORATE SOURCE: Res. Lab. Ciba, Basel, Switzerland
SOURCE: HELVETICA PHYSIOL ET PHARMACOL ACTA, (1961) Vol. 19, No. 3, pp. 335-343.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB Filter paper squares were stained with toluidine blue and after drying treated with heparinic acid producing the metachro-matic red violet. Solutions of antistin and hypertensin and the hydro-chlorides of histamine, **epinephrine**, tryptamine, adenine, d-**glucosamine**, n-octylamine, spermine, privin priscol, mescaline, 256 a and b (the chemical names are given also) and compound 4G/80 as well as the chlorides of NH4, NA, K, Ca and Ma were dropped in the center. Some of the organic substances inhibit the metachromatic effect more or

less. Another series of experiments was conducted with disks of cartilage from calf's ear prepared in a given manner, using the basic dyes toluidine blue and acridine orange. The English summary states: The changes in the metachromatic staining of mucopolysaccharides (poly-electrolytes of the heparin type) which take place upon addition of substances containing basic groups and which as a result of ion exchange reactions are used in three different in vitro systems. When substances of different biological activity are added in equivalent concentration, the dye is released in a quantity dependent on the specific selectivity of a given substance for the active groups of the polyelectrolyte. The displacement of the dye may serve as an indicator for possible liberation mechanisms taking place in similar biological structures. Ascending paper chromatography on heparin-toluidine strips was also used. ABSTRACT AUTHORS: Samuel Amberg

L5 ANSWER 105 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1947:20781 CAPLUS
DOCUMENT NUMBER: 41:20781
ORIGINAL REFERENCE NO.: 41:4179c-e
TITLE: Chemical constitution and inhibition of enzymes; amines inhibiting dihydroxyphenylalanine (dopa) decarboxylase; relation between the latter and amine oxidase
AUTHOR(S): Polonovski, Michel; Schapira, Georges; Gonnard, Pierre
CORPORATE SOURCE: Univ. Paris
SOURCE: Bulletin de la Societe de Chimie Biologique (1946), 28, 735-9
CODEN: BSCIA3; ISSN: 0037-9042
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB cf. C.A. 41, 2785f. Extract of guinea pig kidney in phosphate buffer of pH 7.7 was used as before, and the inhibition of its decarboxylating action on dopa by various amines was studied. For dilute equimol. solns. of the amines the extent of inhibition was, for hordenine, tryptamine, sympathol, arterenol, and benzedrine about 65, for tyramine 60, phenethylamine 47, **ephedrine** and allylamine 45, mescaline 35, diisoallylamine and histamine 28, isoamylamine 25, methylamine 21, **glucosamine**, ethylamine, and putrescine about 15%, and cadaverine and diethylamine about 12%. There was no inhibition by neurine, choline, taurine, aniline, ethanolamine, and amino acids (tyrosine, leucine, phenylalanine, and histidine). All amines having an affinity for amine oxidase inhibit dope decarboxylase. Amines which inhibit dope decarboxylase very little or not at all have no affinity for amine oxidase.

L5 ANSWER 106 OF 106 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1940:18264 CAPLUS
DOCUMENT NUMBER: 34:18264
ORIGINAL REFERENCE NO.: 34:2796d-h
TITLE: Dilituric acid (5-nitrobarbiturates) of some physiologically important bases
AUTHOR(S): Redemann, C. E.; Niemann, Carl
SOURCE: Journal of the American Chemical Society (1940), 62, 590-3
CODEN: JACSAT; ISSN: 0002-7863
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Dilituric acid (I) surpasses picric acid, styphnic acid, picrolonic acid, nitroanilic acid and 3-nitrodiketohydrindene as a reagent for the isolation of many naturally occurring bases. The solubility of I (in millimoles per l.) at 25° is: MeOH 99, 95% EtOH 85, H2O 63, absolute EtOH 35, Me2CO 25, Et2O 0.9, C6H6 0.4. I behaves like a strong monobasic acid, the acid strength being between picric acid and HCl. I is a satisfactory reagent for the separation of K from binary mixts. containing Na and

is an excellent reagent for the isolation of Mg. Solubility of inorg. salts of I in H2O at 25°: Mg 0.1, Ba 1.3, Sr 1.7, Cu 1.8, K 3.9, Ca 5.7, Ag 12, Na 38. Solubility of organic salts of I in H2O at 25°: C2H4(NH2)2 0.6, NH3 7.3, EtNH2 13, Me2NH 15, Ph(iso-Pr)NH 15, BuNH2 17, AmNH2 24, PhNH2 26, MeNH2 37, Bu3N 51, quinoline 23, imidazole 36, C5H5N 54, d(+)-glucobenzimidazole 65, morpholine 80, dl-**ephedrine** 8.7, β-HOC6H4CH2CH2NH2 13, l- **ephedrine** 30, d-pseudoephedrine 32, l-adrenaline 37, HOCH2CH2NH2 53, d-**glucosamine** 85, (HOCH2CH2)3N 132, H2NCH2CH(OH)Me 134, quinine 1.6, brucine 3.7, strychnine 3.9, cinchonine 9.0, nicotine 12.3, caffeine 68, l-diiodotyrosine 2.2, dl- and l-lysine 6.4 and 9.0, l-histidine 7.9, l-tyrosine 10.0, l-arginine 10.2, l-cystine 15, dl-phenylalanine 16, dl-α-aminoisobutyric acid 20, l-tryptophan 20, dl-aminophenylacetic acid 28, sarcosine 34, betaine 34, dl-α-aminobutyric acid 40, l- and dl-alanine 43 and 50, glycine 49, dl- and l-aspartic acid 61 and 94, l- and dl-glutamic acid 67 and 90, dl-γ-aminobutyric acid 77, l-cysteine 92, dl- and l-leucine 94 and 105, l-proline 100, l-asparagine 103, dl-valine 107, dl-isoleucine 107, dl-norleucine 109, dl-norvaline 110, dl-methionine 112, dl-serine 112, l-hydroxyproline 141, guanidine 7.0, tyramine 8.7, histamine 17, phenylethylamine 19, creatinine 23, urea 36. Regeneration of bases from their diliturates can be achieved in the majority of cases by simply replacing the base in question by either C2H4(NH2)2, Mg or NH4; the glycine salt with NH3 gives 89% of crystalline glycine.

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FILE COVERS 1907 - 13 Aug 2007 VOL 147 ISS 8
FILE LAST UPDATED: 12 Aug 2007 (20070812/ED)


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=> glucosamine and (asthma or bronchodilat?)
         22167 GLUCOSAMINE
           327 GLUCOSAMINES
         22270 GLUCOSAMINE
                 (GLUCOSAMINE OR GLUCOSAMINES)
         35967 ASTHMA
            22 ASTHMAS
         35975 ASTHMA
                 (ASTHMA OR ASTHMAS)
          9323 BRONCHODILAT?
L7          49 GLUCOSAMINE AND (ASTHMA OR BRONCHODILAT?)

=> d scan

L7   49 ANSWERS   CAPLUS  COPYRIGHT 2007 ACS on STN
IC   ICM  A61K035-78
     ICS  A61P019-08; A61P019-10; A61P043-00
CC   1-12 (Pharmacology)
     Section cross-reference(s): 18, 63
TI   Use of hyperforin or St. John's wort extracts for the treatment of
     anaphylactic shock and for maintaining and improving bone health
ST   hyperforin bone health anaphylactic shock; St John wort bone health
     anaphylactic shock; hypericum bone health anaphylactic shock
IT   Drug delivery systems
        (aerosols; hyperforin or St. John's wort exts. for treatment of
        anaphylactic shock and for maintaining and improving bone health)
IT   Quaternary ammonium compounds, biological studies
     RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
     (Biological study); USES (Uses)
        (alkylbenzyldimethyl, chlorides; hyperforin or St. John's wort exts.
        for treatment of anaphylactic shock and for maintaining and improving
        bone health)
IT   Psoriasis
        (antipsoriatics; hyperforin or St. John's wort exts. for treatment of
        anaphylactic shock and for maintaining and improving bone health)
IT   Toxins
     RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
     (Biological study); USES (Uses)
        (bee; hyperforin or St. John's wort exts. for treatment of anaphylactic
        shock and for maintaining and improving bone health)
IT   Sulfonic acids, biological studies
     RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
     (Biological study); USES (Uses)
        (bitumen, sodium salts; hyperforin or St. John's wort exts. for
        treatment of anaphylactic shock and for maintaining and improving bone
        health)
IT   Amino acids, biological studies
     RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
     (Biological study); USES (Uses)
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(branched; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(buccal; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(capsules; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(emulsions; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Adrenoceptor agonists
Anaphylaxis
Antiasthmatics
Apoptosis
Bone
Bronchodilators
Dietary supplements
Drug delivery systems
Human
Hypericum
Macrophage
Monocyte
Osteoclast
Platelet (blood)
Polymorphonuclear leukocyte
(hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Leukotrienes
Prostaglandins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Fluorides, biological studies
Natural products, pharmaceutical
Paraffin oils
Vitamins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(inhalants; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(injections, i.m.; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(injections, i.p.; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(injections, i.v.; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(injections, s.c.; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(intraarticular; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(nasal; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)

IT Anti-inflammatory agents
Antirheumatic agents
(nonsteroidal; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(ointments, creams; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(ointments; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(oral; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(rectal; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(solns.; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(suppositories; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(suspensions; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(tablets; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Drug delivery systems
(topical; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT Apoidea
(toxin; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT 59804-37-4, Tenoxicam
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(and other oxicams; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT 13598-36-2D, Phosphonic acid, alkylidenebis- derivs.
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(bisphosphonate; hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT 54397-84-1 71030-39-2, 5-HETE 71160-24-2, LTB4 80619-02-9, 5-Lipoxygenase 329967-85-3, Cyclooxygenase 1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)
IT 50-18-0, Cyclophosphamide 50-21-5, Lactic acid, biological studies 50-33-9, Phenylbutazone, biological studies 50-96-4 52-67-5, D-Penicillamine 53-86-1, Indomethacin 54-05-7, Chloroquine 56-81-5, Glycerin, biological studies 56-85-9, Glutamine, biological studies 57-00-1, Creatine 57-13-6, Urea, biological studies 59-05-2, Methotrexate 59-30-3, Folic acid, biological studies 61-68-7, Mefenamic acid 64-19-7D, Acetic acid, aryl derivs. 68-19-9, Vitamin B12 69-72-7, Salicylic acid, biological studies 69-72-7D, Salicylic acid, derivs. 79-09-4D, Propionic acid, aryl derivs. 97-59-6, Allantoin 107-35-7, Taurine 110-17-8, Fumaric acid, biological studies 112-38-9, Undecylenic acid 118-42-3, Hydroxychloroquine 118-92-3D, Anthranilic acid, derivs. 120-72-9D, Indole, derivs. 129-20-4, Oxyphenbutazone 288-13-1D, Pyrazole, derivs. 298-81-7, Ammoidin

305-03-3, Chlorambucil 446-86-6, Azathioprine 530-78-9, Flufenamic acid 599-79-1, Sulfasalazine 625-08-1 642-72-8, Benzydamine 1143-38-0, Dithranol 1200-22-2, α-Lipoic acid 1306-23-6, Cadmium sulfide, biological studies 1314-13-2, Zinc oxide, biological studies 1406-05-9, Penicillin 1406-16-2, Vitamin D 1406-18-4, Vitamin E 1944-12-3, Fenoterol hydrobromide 2139-47-1, Nifenazone 2210-63-1, Mofebutazone 2438-72-4, Bufexamac 3583-64-0, Bumadizone 4394-00-7, Nifluminic acid 4985-25-5, Pyrazinobutazone 4991-65-5, Tioxolone 5104-49-4 5874-97-5, Orciprenaline sulfate 7440-70-2D, Calcium, salts 7704-34-9, Sulfur, biological studies 9001-54-1, Hyaluronidase 9054-89-1, Orgotein 11079-53-1, Hyperforin 11103-57-4, Vitamin A 12192-57-3 12244-57-4 13055-82-8, Reproterol hydrochloride 13539-59-8, Azapropazone 15307-86-5, Diclofenac 15687-27-1, Ibuprofen 16903-35-8 21898-19-1, Clenbuterol hydrochloride 22071-15-4, Ketoprofen 22204-53-1, Naproxen 22457-89-2, Benfotiamine 22881-35-2, Famprofazone 23031-32-5, Terbutaline sulfate 26171-23-3, Tolmetin 26183-44-8 29031-19-4, **Glucosamine** sulfate 29679-58-1, Fenoprofen 29908-03-0, Ademetionine 30544-47-9, E-Tofenamate 31793-07-4, Pirprofen 33005-95-7, Tiaprofenic acid 33996-33-7, Oxaceprol 34031-32-8, Auranofin 34866-46-1, Carbuterol hydrochloride 36322-90-4, Piroxicam 36330-85-5, Fenbufen 38029-10-6, Pirbuterol hydrochloride 42924-53-8, Nabumetone 51022-70-9, Salbutamol sulfate 53164-05-9, Acemetacin 53716-49-7, Carprofen 53808-88-1, Lonazolac 54350-48-0, Etretinate 56776-01-3, Tulobuterol hydrochloride 57132-53-3, Proglumetacin 62929-91-3, Procaterol hydrochloride 95077-02-4, Serrapeptase
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hyperforin or St. John's wort exts. for treatment of anaphylactic shock and for maintaining and improving bone health)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 49 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
IC ICM A61K039-02
ICS A61K039-04
INCL 424200100; 424248100
CC 15-2 (Immunochemistry)
TI Methods and compounds for the treatment of immunologically-mediated diseases using inactivated and modified Mycobacterium vaccae
ST autoimmune disease vaccine delipidated deglycolipidated Mycobacterium
IT Hydrolysis
(acid; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT T cell (lymphocyte)
(activation; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells in relation to)
IT Allergy
Inflammation
Nose, disease
(allergic rhinitis; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Dermatitis
(atopic; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Infection
(bacterial; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Hydrolysis
(base; methods and compds. for treatment of immune-mediated diseases

using inactivated and modified Mycobacterium vaccae cells)
IT T cell (lymphocyte)
(helper cell/inducer, TH2; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Allergy
(hypersensitivity; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Mycobacterium avium
(infection; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Autoimmune disease
(insulin-dependent diabetes mellitus; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Diabetes mellitus
(insulin-dependent; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Mycobacterium smegmatis
Mycobacterium tuberculosis
(methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium cells)
IT Allergy
Asthma
Atherosclerosis
Eosinophilia
Epithelium
Human
Hypercholesterolemia
Lung, neoplasm
Macrophage
Monocyte
Mycobacterium vaccae
Neoplasm
Respiratory system, disease
Sarcoidosis
Tuberculosis
(methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Interleukins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Interleukin 10
Interleukin 12
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells in relation to formation of)
IT Infection
(mycobacterial; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT Lymphocyte
(natural killer cell, activation; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells in relation to)
IT Glycolipids
Lipids, processes
RL: REM (Removal or disposal); PROC (Process)
(removal; methods and compds. for treatment of immune-mediated diseases

using inactivated and modified Mycobacterium vaccae cells)
IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells in relation to formation of)
IT 7664-39-3, Hydrofluoric acid, biological studies 13444-71-8, Periodic acid 39450-01-6
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(hydrolysis; methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)
IT 50-99-7, Glucose, biological studies 59-23-4, Galactose, biological studies 3416-24-8, **Glucosamine** 3458-28-4, Mannose 7535-00-4, Galactosamine 9036-66-2, Arabinogalactan
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(methods and compds. for treatment of immune-mediated diseases using inactivated and modified Mycobacterium vaccae cells)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 49 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
INCL 424725000; 424765000; 424769000; 435006000; 514002000; 514054000; 514171000
CC 63-4 (Pharmaceuticals)
Section cross-reference(s): 1, 17, 18
TI Composition and method to optimize and customize nutritional supplement formulations by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes
ST nutritional supplement optimization genomics metabolomics diagnosis metab therapy; glyconutrient supplement optimization genomics metabolomics diagnosis metab therapy; nutragenomics diagnosis metab therapy
IT Uncoupling protein
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(2, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Apolipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(A-I, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ABCC8 (ATP-binding cassette transporter subfamily C member 8), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ACE, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to

disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ADRA2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ADRA2A, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ALdB, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ANG, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ANKK1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ANKK1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(APOE, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(AgRP, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Bdnf, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Natural products, pharmaceutical
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Berberis; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Natural products, pharmaceutical
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Bryonia; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(BsmI, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(C825T, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(CART (cocaine- and amphetamine-regulated transcript), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(CART, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(CNR1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(CNRA4, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(CNRA4, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(COMT, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ChREBP, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ChREBP, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Natural products, pharmaceutical
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Chamomlia; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(DAT1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(DBH, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(DRD2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Muscular dystrophy
(Duchenne; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Dopamine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Dopamine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D1A, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Dopamine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D2, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Dopamine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D3, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Dopamine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D4, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis,

metabolism, and therapeutic outcomes)

IT Dopamine receptors

RL: BSU (Biological study, unclassified); BIOL (Biological study) (D5, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Apolipoproteins

RL: BSU (Biological study, unclassified); BIOL (Biological study) (E, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal

RL: BSU (Biological study, unclassified); BIOL (Biological study) (E23K, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT G protein-coupled receptors

RL: BSU (Biological study, unclassified); BIOL (Biological study) (EDG-2 (endothelial differentiation gene 2), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal

RL: BSU (Biological study, unclassified); BIOL (Biological study) (EGR1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal

RL: BSU (Biological study, unclassified); BIOL (Biological study) (ERBB2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal

Gene, animal

RL: BSU (Biological study, unclassified); BIOL (Biological study) (Edg2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Transcription factors

RL: BSU (Biological study, unclassified); BIOL (Biological study) (Egr-1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Garcinia mangostana

(Extract; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Immunophilins

RL: BSU (Biological study, unclassified); BIOL (Biological study) (FKBP (FK 506-binding protein), FKBP5, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Natural products, pharmaceutical

RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological

study); USES (Uses)
(Ferrum Phos; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Fgfr2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Fkbp5, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(FokI, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GAB1 (GRB2-associated binder 1), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GABRA3, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GABRB3, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GFPT1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GFPT2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GLUT-4 (glucose transporter 4), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(GLUT4, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Gab1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Natural products, pharmaceutical
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Gelsemium; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Ghrelin, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HLA-DRB, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Histocompatibility antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HLA-DRB1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HLA-DRB1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Heat-shock proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP 56, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HTR1A, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HTR1D, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HTR2A, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to

disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (HTR2C, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (HTT, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (Hpcall, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study) (ID2 (inhibitor of differentiation 2), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (Id2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (LNC2, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (LNC2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (MAOA, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (MAOA, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (MGPAT, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MGPAT, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Histocompatibility antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MHC (major histocompatibility complex), class II, β chain, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MOG (myelin oligodendrocyte glycoprotein), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MOG, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MOG4, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MOG4, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MTHFR, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Mc3R:UCP1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Mc4R, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NET, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic

outcomes)
IT Glutamate receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NMDA-binding, NMDAR-1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NMDAR1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NYP, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(OBR, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PDGS, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PDGS, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(POMC, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PPARγ, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PTPN1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PTPN22, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Bone, disease

(Paget's; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Blood vessel, disease
(Raynaud's phenomenon; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Arthritis
(Reiter's syndrome; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(SREBP-1 (sterol regulatory element-binding protein 1), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STS, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Sgk, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Sgk1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(TCP-1 (T-complex protein 1), gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(TCP1, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(TDO2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(adiponitrins; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(agouti-related, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Natural products, pharmaceutical
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(aloe; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Disease, animal
(arthropathy; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Mental and behavioral disorders
(attention deficit hyperactivity disorder; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Natural products, pharmaceutical
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(belladonna; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Fats and Glyceridic oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(borage seed; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Cartilage
(bovine; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Neurotrophic factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(brain-derived, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Proteoglycans, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(brevican, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-fos, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-fos, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Transcription factors

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-jun, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-jun, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-myc, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-myc, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Coral
(calcium; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Triterpenes
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(carboxy, boswellic acids; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Shark
(cartilage; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Uncaria tomentosa
(cat's claw; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Fats and Glyceridic oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(currant, Ribes nigrum seed; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Mental and behavioral disorders
(depression; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(desnutrins; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Joint, anatomical
(disease; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease

diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Lipids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(disorders; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(dopamine transporter, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Fats and Glyceridic oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(evening primrose; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Boswellia
Hoodia
Hoodia gordonii
Lagerstroemia speciosa
Momordica charantia
Rhodiola
Sedum roseum
Vaccinium myrtillus
(extract; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Fats and Glyceridic oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fish; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
Aggrecans
Angiotensin AT1 receptors
Cell adhesion molecules
Decorins
GABA receptors
Glucocorticoid receptors
Interleukin 10
Interleukin 1α
Interleukin 1β
Interleukin 8
Leptin receptors
Potassium channel
Ras proteins
Tumor necrosis factors
α2-Adrenoceptors
β-Adrenoceptors
β3-Adrenoceptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Vitamin D receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(genes for, polymorphism in; optimization and customization of

nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Disease, animal
(genetic; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Fats and Glyceridic oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(grape seed; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Tea products
(green; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Tinospora cordifolia
(gulvel; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hippocalcin-like 1, gene for, polymorphism in; optimization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Aconitum
RL: BIOL (Biological study); USES (Uses)
(homeopathic preparation; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Drug delivery systems
(homeopathic; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Chromosome
(human 2, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Collagens, biological studies
Gelatins, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hydrolyzates; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Opioids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Syzygium cumini
(jambolan; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gymnema sylvestre
(leaf; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Embryophyta
(medicinal plant; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Mushroom
(medicinal; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Pituitary hormone receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(melanocortin receptor 3, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Pituitary hormone receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(melanocortin receptor 4, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Disease, animal
(metabolomic; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Perfumes
(myrrh, Guggul; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Disease, animal
(neurogenobolic deficiency syndrome; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Diabetes mellitus
(non-insulin-dependent; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(norepinephrine transporter, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ob, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Resins
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(olibanum, Salai Guggal; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)
IT Acanthopanax senticosus
Alcoholism
Ananas comosus

Anxiety
Appetite depressants
Arthritis
Asthma
Astragalus
Autoimmune disease
Bacopa monnieri
Borago officinalis
Boswellia serrata
Camellia sinensis
Cardiovascular system, disease
Centella asiatica
Citrus aurantium
Commiphora mukul
Curcuma longa
Diabetes mellitus
Diagnosis
Dietary supplements
Dioscorea
Dioscorea villosa
Disease, animal
Drug delivery systems
Drug dependence
Drug toxicity
Echinacea
Flaxseed
Ganoderma lucidum
Genetic polymorphism
Ginkgo
Ginkgo biloba
Harpagophytum procumbens
Human
Hypericum perforatum
Hypertension
Inflammation
Insomnia
Larix
Lentinula edodes
Ligustrum
Linum usitatissimum
Lupus erythematosus
Lyme disease
Magnolia
Metabolism, animal
Mutation
Neoplasm
Obesity
Oenothera
Oenothera biennis
Osteoarthritis
Osteoporosis
Oxidative stress, biological
Pain
Panax
Panax ginseng
Panax quinquefolium
Parthenium hysterophorus
Passiflora
Perna canaliculus
Perna viridis
Phellodendron amurense
Piper methysticum

Primula
Pterocarpus marsupium
Rheumatic fever
Rheumatoid arthritis
Rhododendron
Ribes nigrum
Salix alba
Schisandra
Simulation and Modeling
Sleep disorders
Stress, animal
Tabebuia
Therapy
Tonga
Trigonella foenum-graecum
Tripterygium wilfordii
Urtica dioica
Valeriana
Valeriana officinalis
Zingiber officinale
(optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT DNA
RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Carbohydrate metabolism disorders
Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Amino acids, biological studies
Collagens, biological studies
Gelatins, biological studies
Linseed oil
Mineral elements, biological studies
Monosaccharides
Natural products, pharmaceutical
Oligosaccharides, biological studies
Polysaccharides, biological studies
Tannins
Vitamins
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Tobacco smoke
(overcoming dependence of; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Rheumatic diseases
(palindromic; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Mental and behavioral disorders
(post-traumatic stress disorder; optimization and customization of

nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Ovarian cycle
(premenstrual syndrome; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Hair
Mental activity
Nail (anatomical)
Skin
(promotion of health of; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Arthritis
(reactive; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Vitis vinifera
(seeds; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Genetic polymorphism
(single nucleotide; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(sortilin-1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Neurotransmitters
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(synthesis precursors; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Carboxylic acids, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(triterpene, boswellic acids; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Neurotensin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Fibroblast growth factor receptors
Neurotensin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 2, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT1A, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT1D, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT2A, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT2B, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT2C, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT4, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 5-HT receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 5-HT7, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Cannabinoid receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type CB1, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Collagens, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(type II; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Blood vessel, disease
Inflammation
(vasculitis, hypersensitivity; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α, gene for, polymorphism in; optimization and customization of

nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT α2-Adrenoceptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α2A-, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β3AR, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ2, gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 54-11-5, Nicotine
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(dependence to and treatment with; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 17031-92-4, Calcium pyrophosphate dihydrate
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(disorder; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 9000-83-3, ATPase 9012-25-3, Catechol O-methyltransferase 9013-38-1, Dopamine β-hydroxylase 9014-51-1, Tryptophan 2,3-dioxygenase 9015-82-1, Angiotensin converting enzyme 9025-62-1, Steroid sulfatase 9028-86-8, Aldehyde dehydrogenase 9029-96-2, Glycerol 3-phosphate acyltransferase 9030-45-9, Glutamine-fructose 6-phosphate amidotransferase 9031-91-8, **Glucosamine** 6-phosphate acetyltransferase 9035-51-2, Cytochrome P 450, biological studies 9041-46-7, 11β-Hydroxysteroid dehydrogenase, type 1 66796-54-1, Proopiomelanocortin 71822-25-8, Methylene tetrahydrofolate reductase 82785-45-3, Neuropeptide Y 88402-55-5, Prodynorphin 90680-95-8, Proenkephalin 178037-70-2, Protein kinase SGK-1 300865-11-6, Protein tyrosine phosphatase PTPN1 301156-78-5, Protein tyrosine phosphatase Lyp 304853-26-7, Ghrelin 330597-62-1, CYP2D6
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(gene for, polymorphism in; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 9000-81-1, Acetylcholinesterase 9001-08-5, Cholinesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 51-84-3, Acetylcholine, biological studies 39379-15-2, Neurotensin
RL: BSU (Biological study, unclassified); FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 50-81-7, Vitamin C, biological studies 50-99-7, D-Glucose, biological studies 53-43-0, DHEA 54-47-7, Pyridoxal-5'-phosphate 56-85-9, L-Glutamine, biological studies 56-86-0, L-Glutamic acid, biological studies 58-86-6, Xylose, biological studies 59-23-4, Galactose, biological studies 59-30-3, Folic acid, biological studies 59-43-8, Thiamine, biological studies 59-92-7, biological studies 60-18-4, L-Tyrosine, biological studies 62-49-7, Choline 63-91-2, L-Phenylalanine, biological studies 66-84-2, **Glucosamine** hydrochloride 67-68-5, DMSO, biological studies 67-71-0, Methylsulfonylmethane 73-22-3, L-Tryptophan, biological studies 73-31-4, Melatonin 94-07-5, Synephrine 117-39-5, Quercetin 131-48-6, N-Acetylneuraminic acid 147-81-9, Arabinose 150-30-1, DLPA 299-42-3, Ephedrine 300-62-9D, Amphetamine, derivs. 328-38-1, D-Leucine 501-52-0, Hydrocinnamic acid 506-26-3, γ-Linolenic acid 541-15-1, L-Carnitine 673-06-3, D-Phenylalanine 989-51-5, EGCG 1406-18-4, Vitamin E 1811-31-0, N-Acetylgalactosamine 2438-80-4, Fucose 3416-24-8, **Glucosamine** 3458-28-4, D-Mannose 4350-09-8, 5-Hydroxytryptophan 7439-95-4, Magnesium, biological studies 7439-96-5, Manganese, biological studies 7440-09-7, Potassium, biological studies 7440-47-3D, Chromium, salts 7440-70-2, Calcium, biological studies 7512-17-6, N-Acetylglucosamine 7693-13-2, Calcium citrate 7778-49-6, Potassium citrate 7779-25-1, Magnesium citrate 8059-24-3, Vitamin B6 9001-73-4, Papain 9004-61-9, Hyaluronic acid 9007-28-7, Chondroitin sulfate 9036-66-2, Arabinogalactan 12001-76-2, Vitamin B complex 13539-59-8, Cinnamin 14007-45-5, Potassium aspartate 16351-10-3, Manganese ascorbate 18962-61-3, Magnesium aspartate 21059-46-1, Calcium aspartate 27750-10-3, (-)-Hydroxycitric acid 27750-10-3D, (-)-Hydroxycitric acid, salts 27774-13-6, Vanadyl sulfate 29031-19-4, Glucosamine sulfate 29908-03-0, S-Adenosyl-L-methionine 31271-07-5, γ-Mangostin 39345-92-1, Chromium chloride 40816-51-1 64660-84-0, Cetyl-M 102518-79-6, Huperzine A 140947-78-0 150977-36-9, Bromelain 316129-64-3, Protykin 880087-81-0 880150-86-7, Boswellin 880150-96-9, Sierrasil 880150-98-1, AlgaeCal 880151-04-2, Synaptamine 880260-05-9, Synaptose
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

IT 50-67-9, Serotonin, biological studies 51-61-6, Dopamine, biological studies 56-12-2, γ-Aminobutyric acid, biological studies 3040-38-8, Acetylcarnitine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(precursors; optimization and customization of nutritional supplement by measuring genetic and metabolomic contributing factors to disease diagnosis, stratification, prognosis, metabolism, and therapeutic outcomes)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 49 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
CC 15-2 (Immunochemistry)
TI An epitope residing in carbohydrate chains of a sea squirt antigen termed Gi-rep
ST Styela antigen carbohydrate epitope
IT Styela plicata
(antigen of, epitope on carbohydrate chains of)

IT Oligosaccharides
RL: BIOL (Biological study)
(antigenic determinant on, of sea squirt antigen)
IT Allergens
Antigens
RL: BIOL (Biological study)
(determinant, of sea squirt)
IT Amino acids, biological studies
Carbohydrates and Sugars, biological studies
RL: BIOL (Biological study)
(of antigen from sea squirt)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 49 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
IC ICM A61K031-737
ICS A61K031-42; A61K031-501; A61K031-415; A61P029-00; A61K031-737; A61K031-42; A61K031-737; A61K031-50; A61K031-737; A61K031-501; A61K031-737; A61K031-415
CC 1-7 (Pharmacology)
Section cross-reference(s): 63
TI Compositions for the treatment and prevention of pain and inflammation with a cyclooxygenase-2 selective inhibitor and chondroitin sulfate
ST chondroitin sulfate cyclooxygenase 2 inhibitor analgesic antiinflammatory; pain inflammation chondroitin sulfate COX2 inhibitor **glucosamine**
IT Inflammation
(Crohn's disease; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Intestine, disease
(Crohn's; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Swelling, biological
(after injury; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Anemia (disease)
(aplastic; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Bronchi, disease
Inflammation
(bronchitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Joint, anatomical
(bursa, bursitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Mycosis
(candidiasis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Ischemia
(cardiac; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Intestine, neoplasm
(colorectal; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Eye, disease
Inflammation
(conjunctivitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Menstrual disorder
(cramps; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT AIDS (disease)
Alzheimer's disease

Analgesics
Anti-AIDS agents
Anti-Alzheimer's agents
Anti-inflammatory agents
Anti-ischemic agents
Antiarthritics
Antiasthmatics
Antidiabetic agents
Antipyretics
Antirheumatic agents
Antitumor agents
Antiulcer agents
Arthritis
Asthma
Behcet's syndrome
Blood vessel, disease
Burn
Calculi, renal
Cardiovascular agents
Connective tissue, disease
Dermatitis
Digestive tract, disease
Drug delivery systems
Eczema
Eye, disease
Fever and Hyperthermia
Gastrointestinal agents
Gout
Headache
Hodgkin's disease
Human
Human herpesvirus
Human immunodeficiency virus
Inflammation
Multiple sclerosis
Myasthenia gravis
Neoplasm
Nervous system, disease
Nervous system agents
Osteoarthritis
Pain
Psoriasis
Rheumatic fever
Rheumatoid arthritis
Sarcoidosis
Skin, disease
Wound
Wound healing promoters
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Mental and behavioral disorders
(dementia, cortical; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Dentistry
(dental pain; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Tendon
(disease, tendinitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Lung, disease
(edema; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Drug delivery systems
(enteric; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Stomach, disease
(gastric varices; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Ulcer
(gastric; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Inflammation
Stomach, disease
(gastritis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Gingiva, disease
Inflammation
(gingivitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Antiviral agents
(herpes simplex infection; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Allergy
(hypersensitivity; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Intestine, disease
(inflammatory; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Connective tissue
Eye, disease
(injury; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Autoimmune disease
(insulin-dependent diabetes mellitus; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Diabetes mellitus
(insulin-dependent; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Intestine, disease
(irritable bowel syndrome; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Heart, disease
(ischemia; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Arthritis
(juvenile; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Spinal column, disease
(lumbar spondylanhrosis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Spinal column, disease
(lumbar spondylarthrosis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Headache
(migraine; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Injury
(minor; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Kidney, disease
(nephrotic syndrome; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Injury

(ocular; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Drug delivery systems
(parenterals; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Artery, disease
Inflammation
(periarteritis nodosa; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Eye, disease
(photophobia; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Inflammation
Lung, disease
(pneumonitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Myositis
(polymyositis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Drug delivery systems
(prodrugs; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Edema
(pulmonary; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Eye, disease
Inflammation
(retinitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Eye, disease
(retinopathy; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Connective tissue, disease
(scleroderma; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Headache
(sinus; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Muscle, disease
(spasm, menstrual cramps; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Spinal column, disease
(spondyloarthropathy; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Lupus erythematosus
(systemic; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Inflammation
(tendinitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Headache
(tension; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Inflammation
Thyroid gland, disease
(thyroiditis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Stomach, disease
(ulcer; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)
IT Inflammation
Intestine, disease

(ulcerative colitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Eye, disease
Inflammation
(uveitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT Inflammation
Vagina, disease
(vaginitis; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT 220991-20-8, COX 189 220991-20-8D, COX 189, prodrug derivs.
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(COX 189; cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT 329900-75-6, Cyclooxygenase 2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT 103-82-2D, Phenylacetic acid, derivs. 254-04-6D, 2H-1-Benzopyran, derivs. 254-37-5D, 2H-1-Benzothiopyran, derivs. 493-08-3D, Chroman, derivs. 528-04-1 1398-61-4D, Chitin, hydrolysis products and derivs., **glucosamine**-containing 1746-32-3, N-Acetylglucosamine-6-phosphate 3616-42-0, **Glucosamine**-6-phosphate 4607-22-1 7512-17-6, N-Acetyl-D-**glucosamine** 9004-61-9D, Hyaluronic acid, **glucosamine**-containing 9005-49-6D, Heparin, **glucosamine**-containing, biological studies 9007-28-7, Chondroitin sulfate 9056-36-4D, Keratosulfate, **glucosamine**-containing 19792-88-2, **Glucosamine**-2,6-disulfate 19889-76-0, **Glucosamine**-1-phosphate 28446-21-1, N-Acetylglucosamine-1-phosphate 29828-28-2D, Dihydronaphthalene, derivs. 29968-14-7D, Dihydroquinoline, derivs. 53942-44-2 71125-38-7, Meloxicam 71125-38-7D, Meloxicam, prodrug derivs. 76330-20-6 76330-21-7 78794-60-2 78794-60-2D, prodrug derivs. 91674-26-9, **Glucosamine**-6-sulfate 93014-16-5 93014-16-5D, prodrug derivs. 98094-70-3 143809-38-5 143809-38-5D, prodrug derivs. 143809-39-6 143809-39-6D, prodrug derivs. 158959-32-1 158959-32-1D, prodrug derivs. 158959-33-2 158959-33-2D, prodrug derivs. 158959-34-3 158959-34-3D, prodrug derivs. 158959-35-4 158959-35-4D, prodrug derivs. 158959-37-6 158959-37-6D, prodrug derivs. 158959-42-3 158959-42-3D, prodrug derivs. 158959-43-4 158959-43-4D, prodrug derivs. 158959-46-7 158959-46-7D, prodrug derivs. 158959-47-8 158959-47-8D, prodrug derivs. 158959-56-9 158959-56-9D, prodrug derivs. 159429-69-3 159429-69-3D, prodrug derivs. 159429-70-6 159429-70-6D, prodrug derivs. 162011-64-5 162011-64-5D, prodrug derivs. 162011-90-7, Rofecoxib 162011-90-7D, Rofecoxib, prodrug derivs. 162054-19-5 162054-19-5D, prodrug derivs. 163303-19-3 163303-19-3D, prodrug derivs. 163303-25-1 163303-25-1D, prodrug derivs. 163303-29-5 163303-29-5D, prodrug derivs. 163303-38-6 163303-38-6D, prodrug derivs. 163303-55-7 163303-55-7D, prodrug derivs. 165252-03-9 165252-03-9D, prodrug derivs. 165328-42-7 165328-42-7D, prodrug derivs. 165328-49-4 165328-49-4D, prodrug derivs. 165328-51-8 165328-51-8D, prodrug derivs. 165328-52-9 165328-52-9D, prodrug derivs. 168299-83-0 168299-83-0D, prodrug derivs. 168299-90-9 168299-90-9D, prodrug derivs. 168433-84-9 168433-84-9D, prodrug derivs. 169154-04-5 169154-04-5D, prodrug derivs. 169154-07-8 169154-07-8D, prodrug derivs. 169154-19-2 169154-19-2D, prodrug derivs. 169154-24-9 169154-24-9D, prodrug derivs. 169590-41-4 169590-41-4D, prodrug derivs. 169590-42-5 169590-42-5D, prodrug derivs. 169902-71-0 169902-71-0D, prodrug derivs. 169902-74-3 169902-74-3D, prodrug derivs. 169902-75-4 169902-75-4D, prodrug derivs. 169951-23-9 169951-23-9D, prodrug derivs. 169951-24-0 169951-24-0D,

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170569-87-6 170569-87-6D, prodrug derivs. 170569-88-7 170569-88-7D,
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170570-05-5 170570-05-5D, prodrug derivs. 170570-25-9 170570-25-9D,
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170571-71-8 170571-71-8D, prodrug derivs. 171888-46-3 171888-46-3D,
prodrug derivs. 173776-67-5 173776-67-5D, prodrug derivs.
174454-52-5 174454-52-5D, prodrug derivs. 175676-91-2 175676-91-2D,
prodrug derivs. 175676-92-3 175676-92-3D, prodrug derivs.
175677-05-1 175677-05-1D, prodrug derivs. 175677-13-1 175677-13-1D,
prodrug derivs. 175677-14-2 175677-14-2D, prodrug derivs.
175679-48-8 175679-48-8D, prodrug derivs. 175679-49-9 175679-49-9D,
prodrug derivs. 175883-05-3 175883-05-3D, prodrug derivs.
175883-36-0 175883-36-0D, prodrug derivs. 177560-19-9 177560-19-9D,
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177660-89-8 177660-89-8D, prodrug derivs. 177660-92-3 177660-92-3D,
prodrug derivs. 177660-94-5 177660-94-5D, prodrug derivs.
177661-00-6 177661-00-6D, prodrug derivs. 177661-01-7 177661-01-7D,
prodrug derivs. 177661-02-8 177661-02-8D, prodrug derivs.
177661-04-0 177661-04-0D, prodrug derivs. 177661-06-2 177661-06-2D,
prodrug derivs. 177661-15-3 177661-15-3D, prodrug derivs.
177661-17-5 177661-17-5D, prodrug derivs. 177661-18-6 177661-18-6D,
prodrug derivs. 177661-19-7 177661-19-7D, prodrug derivs.
177661-49-3 177661-49-3D, prodrug derivs.
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and prevention of pain and inflammation)

IT 177662-22-5 177662-22-5D, prodrug derivs. 177754-42-6 177754-42-6D,
prodrug derivs. 177755-10-1 177755-10-1D, prodrug derivs.
178402-36-3 178402-36-3D, prodrug derivs. 179382-91-3 179382-91-3D,
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180200-68-4, JTE-522 180200-68-4D, JTE-522, prodrug derivs.
181627-94-1 181627-94-1D, prodrug derivs. 181627-96-3 181627-96-3D,
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181628-00-2 181628-00-2D, prodrug derivs. 181695-72-7 181695-72-7D,
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181695-85-2 181695-85-2D, prodrug derivs. 181696-18-4 181696-18-4D,
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181809-58-5 181809-58-5D, prodrug derivs. 181809-60-9 181809-60-9D,
prodrug derivs. 181809-63-2 181809-63-2D, prodrug derivs.
183610-65-3 183610-65-3D, prodrug derivs. 185344-51-8 185344-51-8D,
prodrug derivs. 185344-55-2 185344-55-2D, prodrug derivs.
186804-93-3 186804-93-3D, prodrug derivs. 198470-84-7, Parecoxib

198470-84-7D, Parecoxib, prodrug derivs. 202409-33-4, Etoricoxib
202409-33-4D, Etoricoxib, prodrug derivs. 215122-07-9 215122-07-9D,
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215122-44-4 215122-44-4D, prodrug derivs. 215122-45-5 215122-45-5D,
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215122-48-8 215122-48-8D, prodrug derivs. 215122-49-9 215122-49-9D,
prodrug derivs. 215122-50-2 215122-50-2D, prodrug derivs.
215122-51-3 215122-51-3D, prodrug derivs. 215122-52-4 215122-52-4D,
prodrug derivs. 215122-53-5 215122-53-5D, prodrug derivs.
215122-55-7 215122-55-7D, prodrug derivs. 215122-56-8 215122-56-8D,
prodrug derivs. 215122-58-0 215122-58-0D, prodrug derivs.
215122-59-1 215122-59-1D, prodrug derivs. 215122-60-4 215122-60-4D,
prodrug derivs. 215122-61-5 215122-61-5D, prodrug derivs.
215122-62-6 215122-62-6D, prodrug derivs. 215122-63-7 215122-63-7D,
prodrug derivs. 215122-65-9 215122-65-9D, prodrug derivs.
215122-69-3 215122-69-3D, prodrug derivs. 215122-70-6 215122-70-6D,
prodrug derivs. 215122-71-7 215122-71-7D, prodrug derivs.
215122-75-1 215122-75-1D, prodrug derivs. 215122-76-2 215122-76-2D,
prodrug derivs. 215122-77-3 215122-77-3D, prodrug derivs.
215123-07-2 215123-07-2D, prodrug derivs. 215123-08-3 215123-08-3D,
prodrug derivs. 215123-16-3 215123-16-3D, prodrug derivs.
215123-80-1 215123-80-1D, prodrug derivs. 215123-84-5 215123-84-5D,
prodrug derivs. 266320-83-6 266320-83-6D, prodrug derivs.
485384-62-1 485384-62-1D, prodrug derivs. 499764-05-5 499770-50-2
499770-51-3
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and
prevention of pain and inflammation)

IT 122-00-9, 4'-Methylacetophenone 383-63-1, Ethyl trifluoroacetate
4392-54-5, 4-Sulfonamidophenylhydrazine
RL: RCT (Reactant); RACT (Reactant or reagent)
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and
prevention of pain and inflammation)

IT 720-94-5P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT
(Reactant or reagent)
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and
prevention of pain and inflammation)

IT 66-84-2, **Glucosamine** hydrochloride 3416-24-8,
Glucosamine 3416-24-8D, **Glucosamine**, acid salts
15167-91-6 29031-19-4, **Glucosamine** sulfate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(cyclooxygenase 2 inhibitor and chondroitin sulfate for treatment and
prevention of pain and inflammation, and use with **glucosamine**
)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 49 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
IC ICM A23K001-165
INCL 424442000
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 18
TI Oral administration form for veterinary use containing oils
ST oral veterinary compn oil; worm remover pyrantel pamoate mineral oil
IT Fatty acids, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(esters; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Chamomile
Echinacea
Foeniculum vulgare
(exts.; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Alcohols, biological studies
Esters, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fatty; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Drug delivery systems
(homeopathic; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Fats and Glyceridic oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hydrogenated; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Palm oil
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hydrogenated; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Joint, anatomical
(medical care of; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Drug delivery systems
(oral; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Worm
(removing of; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Fats and Glyceridic oils, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(vegetable, hydrogenated; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Analgesics
Antibiotics
Antidiarrheals
Antihistamines
Antirheumatic agents
Beeswax
Bronchodilators
Canis familiaris
Diuretics
Felis catus
Pet animal
Radiopharmaceuticals
(veterinary oral compns. containing active ingredients and oils and thickening agents)

IT Minerals, biological studies
Soybean oil
Vitamins
Waxes
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Castor oil
Corn oil
Hormones, animal, biological studies
Hydrocarbon oils
Kaolin, biological studies
Oils
Peanut oil
Polyoxyalkylenes, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(veterinary oral compns. containing active ingredients and oils and thickening agents)
IT Dietary supplements
(vitamins for animals; veterinary oral compns. containing active ingredients and oils and thickening agents)
IT 50-81-7, Vitamin C, biological studies 59-43-8, Thiamine, biological studies 67-97-0, Vitamin D3 98-92-0, Niacinamide 471-34-1, Calcium carbonate, biological studies 1406-18-4, Vitamin E 11103-57-4, Vitamin A
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(veterinary oral compns. containing active ingredients and oils and thickening agents)
IT 121-79-9, Propyl gallate 128-37-0, Butylated Hydroxy toluene, biological studies 9004-34-6, Cellulose, biological studies 22204-24-6, Pyrantel pamoate 25322-68-3, PEG 26787-78-0, Amoxycillin 29031-19-4, **Glucosamine** sulfate 31566-31-1, Glyceryl monostearate 36653-82-4, Cetyl alcohol 37148-27-9, Clenbuterol
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(veterinary oral compns. containing active ingredients and oils and thickening agents)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 49 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
IC ICM A61K031-192
ICS A61K031-485; A61P025-04
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1
TI Non-steroidal antiinflammatory drug and **glucosamine** combination
ST nonsteroidal antiinflammatory drug **glucosamine**
IT Opioids
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(analgesics; nonsteroidal antiinflammatory drug and **glucosamine** combination)
IT Analgesics
Antiarthritics
Antihistamines
Bronchodilators
Decongestants
Drug delivery systems
Human
Hypnotics and Sedatives
Muscle relaxants
Pain

(nonsteroidal antiinflammatory drug and **glucosamine** combination)
IT Anti-inflammatory agents
(nonsteroidal; nonsteroidal antiinflammatory drug and **glucosamine** combination)
IT Drug interactions
(synergistic; nonsteroidal antiinflammatory drug and **glucosamine** combination)
IT Drugs
(veterinary; nonsteroidal antiinflammatory drug and **glucosamine** combination)
IT 66-84-2, **Glucosamine** hydrochloride 3416-24-8, **Glucosamine** 6490-70-6, α- **Glucosamine** 7512-17-6, N-Acetylglucosamine 14257-69-3, β- **Glucosamine** 15687-27-1, Ibuprofen 22071-15-4, Ketoprofen 29031-19-4, **Glucosamine** sulfate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nonsteroidal antiinflammatory drug and **glucosamine** combination)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> d his

(FILE 'HOME' ENTERED AT 15:08:54 ON 13 AUG 2007)

FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 15:09:36 ON 13 AUG 2007
L1 12 S (GLUCOSAMIN? OR MANNOSAMIN?) AND (SAMBUTEROL OR BITOLTEROL OR
L2 129 S (GLUCOSAMIN? OR MANNOSAMIN?) AND (DOPEXAMINE OR EPHEDRINE OR
L3 6 S (GLUCOSAMIN? OR MANNOSAMIN?) AND (ISOETARINE OR ISOPRETERENOL
L4 9 S (GLUCOSAMIN? OR MANNOSAMIN?) AND (RITODRINE OR SALBUTAMOL OR
L5 106 DUP REM L1-L4 (50 DUPLICATES REMOVED)
L6 82 S L5 AND PY<=2003

FILE 'STNGUIDE' ENTERED AT 15:15:17 ON 13 AUG 2007

FILE 'CAPLUS' ENTERED AT 15:30:32 ON 13 AUG 2007
L7 49 GLUCOSAMINE AND (ASTHMA OR BRONCHODILAT?)

=> l7 and Non-steroidal antiinflammatory drug and glucosamine combination/ti
895335 NON
34 NONS
895362 NON
(NON OR NONS)
22363 STEROIDAL
5 STEROIDALS
22364 STEROIDAL
(STEROIDAL OR STEROIDALS)
51665 ANTIINFLAMMATORY
1006 ANTIINFLAMMATORIES
51825 ANTIINFLAMMATORY
(ANTIINFLAMMATORY OR ANTIINFLAMMATORIES)
749194 DRUG
335816 DRUGS
909251 DRUG
(DRUG OR DRUGS)
506 NON-STEROIDAL ANTIINFLAMMATORY DRUG
(NON(W)STEROIDAL(W)ANTIINFLAMMATORY(W)DRUG)
3216 GLUCOSAMINE/TI
62 GLUCOSAMINES/TI
3276 GLUCOSAMINE/TI
((GLUCOSAMINE OR GLUCOSAMINES)/TI)

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        41636 COMBINATION/TI
         9461 COMBINATIONS/TI
        51051 COMBINATION/TI
                ((COMBINATION OR COMBINATIONS)/TI)
            1 GLUCOSAMINE COMBINATION/TI
                ((GLUCOSAMINE(W)COMBINATION)/TI)
L8          1 L7 AND NON-STEROIDAL ANTIINFLAMMATORY DRUG AND GLUCOSAMINE
              COMBINATION/TI

=> d 18 ibib abs

L8   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2006:228050  CAPLUS
DOCUMENT NUMBER:         144:318552
TITLE:                   Non-steroidal
                         antiinflammatory drug and
                         glucosamine combination
INVENTOR(S):             Salama, Jean-Pierre
PATENT ASSIGNEE(S):      Jean-Pierre Salama, Australia
SOURCE:                  Aust. Pat. Appl., 6 pp.
                         CODEN: AUXXCM
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     ----------------    ----   --------    --------------------   --------
     AU 2003203346       A1     20041021    AU 2003-203346         20030330
PRIORITY APPLN. INFO.:                      AU 2003-203346         20030330
AB   The invention relates to a composition comprising a glucosamine
     material and an analgesic such as an a nonsteroidal antiinflammatory drug
     and/or opioid analgesic and its use for treatment of pain in
     pharmaceutical or veterinary applications. When the components of the
     compns. are administered within certain ratios, the analgesic efficacy of
     the composition is super additive (synergistic) relative to the analgesic
     efficacy of the analgesic compound alone.


=> s l7 and py<=2002
      22880538 PY<=2002
L9          16 L7 AND PY<=2002

=> d l9 1-16 ibib abs

L9   ANSWER 1 OF 16  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:1060506  CAPLUS
DOCUMENT NUMBER:         142:36910
TITLE:                   Methods and compounds for the treatment of
                         immunologically-mediated diseases using inactivated
                         and modified Mycobacterium vaccae
INVENTOR(S):             Watson, James D.; Tan, Paul L. J.; Prestidge, Ross L.;
                         Abernethy, Nevin
PATENT ASSIGNEE(S):      Genesis Research and Development Corporation Limited,
                         N. Z.
SOURCE:                  U.S. Pat. Appl. Publ., 32 pp., Cont.-in-part of U.S.
                         Ser. No. 710,425.
                         CODEN: USXXCO
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:
```

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2004247622	A1	20041209	US 2004-825709		20040416	
US 6350457	B1	20020226	US 1999-449013		19991124	<--
US 6723327	B1	20040420	US 2000-710425		20001108	
PRIORITY APPLN. INFO.:			US 1999-137112P	P	19990602	
			US 1999-449013	A2	19991124	
			US 2000-710425	A2	20001108	
			WO 2000-NZ85	A	20000601	

AB Methods for the prevention and treatment of disorders, including disorders of the skin and respiratory system, such as infection with mycobacteria (M. tuberculosis or M. avium), sarcoidosis, **asthma**, allergic rhinitis, allergic dermatitis, and lung cancers are provided. The methods comprise administering a composition comprising at least one derivative of delipidated and deglycolipidated M. vaccae cells. The delipidated and deglycolipidated M. tuberculosis and M. smegmatis cells, and their derivs. can also be used.

L9 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:327155 CAPLUS
DOCUMENT NUMBER: 140:337908
TITLE: Methods and compounds for the treatment of immunologically mediated diseases using Mycobacterium vaccae
INVENTOR(S): Watson, James D.; Tan, Paul L. J.; Prestidge, Ross; Abernethy, Nevin
PATENT ASSIGNEE(S): Genesis Research and Development Corporation, N. Z.
SOURCE: U.S., 28 pp., Cont.-in-part of U.S. Ser. No. 449,013.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 6723327	B1	20040420	US 2000-710425		20001108	
US 6350457	B1	20020226	US 1999-449013		19991124	<--
WO 2000074715	A1	20001214	WO 2000-NZ85		20000601	<--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM						
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG						
US 2004247622	A1	20041209	US 2004-825709		20040416	
PRIORITY APPLN. INFO.:			US 1999-137112P	P	19990602	
			US 1999-449013	A2	19991124	
			WO 2000-NZ85	A	20000601	
			US 2000-710425	A2	20001108	

AB Methods for the prevention and treatment of disorders, including disorders of the skin and respiratory system, such as infection with mycobacteria such as M. tuberculosis or M. avium, sarcoidosis, **asthma**, allergic rhinitis, allergic dermatitis and lung cancers are provided, such methods comprising administering a composition comprising at least one derivative of delipidated and deglycolipidated M. vaccae cells.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:892670 CAPLUS
DOCUMENT NUMBER: 139:366867
TITLE: Preparation of small crystals
INVENTOR(S): McCausland, Linda Jane; Reay, David
PATENT ASSIGNEE(S): Accentus PLC, UK
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003092851	A1	20031113	WO 2003-GB1540	20030408
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002089942	A1	20021114	WO 2002-GB2006	20020502 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003224261	A1	20031117	AU 2003-224261	20030408
EP 1499410	A1	20050126	EP 2003-720684	20030408
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 2005155541	A1	20050721	US 2003-513153	20030408
JP 2005524519	T	20050818	JP 2004-501028	20030408
PRIORITY APPLN. INFO.:			WO 2002-GB2006	A 20020502
			GB 2002-19815	A 20020824
			GB 2001-11083	A 20010505
			GB 2001-27380	A 20011115
			WO 2003-GB1540	W 20030408

AB Small crystals, e.g., of proteins, sugars, or drugs, are made by mixing a solution of a desired substance with an antisolvent in a fluidic vortex mixer in which the residence time is <1 s, e.g., 10 ms. The liquid in the fluidic vortex mixer is subjected to high intensity ultrasound from a transducer. The solution very rapidly becomes supersatd., and the ultrasound can induce a very large number of nuclei for crystal growth. Small crystals, e.g., <5 μm, are formed. The resulting suspension is treated to add or remove ingredients, and then spray dried using an atomizer tuned to create small droplets in such a way that each droplet should contain not more than one crystal. Crystal agglomeration is prevented.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:657870 CAPLUS
DOCUMENT NUMBER: 138:198025
TITLE: Proteoglycans in inflammation
AUTHOR(S): Delehedde, M.; Allain, F.; Payne, S. J.; Borgo, R.; Vanpouille, C.; Fernig, D. G.; Deudon, E.
CORPORATE SOURCE: School of Biological Sciences, University of Liverpool, Liverpool, L69 7ZB, UK
SOURCE: Current Medicinal Chemistry: Anti-Inflammatory & Anti-Allergy Agents (2002), 1(2), 89-102
CODEN: CMCAGM; ISSN: 1568-0142
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Proteoglycans (PG) consist of a core protein and an associated glycosaminoglycan (GAG) chain and reside on the cell surface and in the extracellular matrix. The different GAG chains of PG, heparan sulfate/heparin (HS), dermatan/chondroitin sulfate, keratan sulfate and of hyaluronic acid, which is not associated with a core protein, are synthesized as polymers of repeating disaccharide units. However, the structures of GAG chains are highly diverse. For example, the post-polymerization modification of heparan chains (a polymer of glucuronic acid β1-4 N-acetyl glucosamine) by the sulfation of specific residues and the epimerization of glucuronate to iduronate generates HS, which has a potential sequence complexity greater than that of the human proteome. Although only a fraction of this chemical complexity is used, it provides the framework for GAG chains to interact with a vast repertoire of proteins, with a specificity that is as high as required. As a consequence of their multiple interactions, PG are intimately involved in the different stages of inflammation, from the recruitment of inflammatory cells to the release of mediators of inflammation by infiltrating leukocytes and the turnover of extracellular matrix. The overarching theme of PG in inflammation is the regulation of the inflammatory microenvironment, which must change continuously and dynamically during the progression of the inflammatory response as observed in various pathologies such as arthritis and asthma. These changes include the modulation of the activity of GAG-binding cytokines, growth factors, proteases and protease inhibitors. The interactions of these regulatory proteins with GAG provides much of the focus for GAG-based therapeutic targets.
REFERENCE COUNT: 182 THERE ARE 182 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:256052 CAPLUS
DOCUMENT NUMBER: 136:284456
TITLE: Analgesic and glucosamine compositions
INVENTOR(S): Raffa, Robert; Cowan, Alan; Tallarida, Ronald
PATENT ASSIGNEE(S): Temple University, USA
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002026239	A1	20020404	WO 2001-US29606	20010921 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,				

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2423585	A1	20020404	CA 2001-2423585		20010921 <--
AU 2001092929	A5	20020408	AU 2001-92929		20010921 <--
EP 1328278	A1	20030723	EP 2001-973339		20010921
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
US 2002058642	A1	20020516	US 2001-964178		20010925 <--
US 6900189	B2	20050531			
PRIORITY APPLN. INFO.:			US 2000-235405P	P	20000926
			WO 2001-US29606	W	20010921

AB This invention relates to a composition a **glucosamine** material and an analgesic compound such as a nonsteroidal anti-inflammatory drug (NSAID) and/or an opioid analgesic and its use for treatment of pain in pharmaceutical or veterinary applications. When the components are administered within certain ratios, the analgesic efficacy of the composition is super-additive (synergistic) relative to the analgesic efficacy of the analgesic compound alone. Solns. of **glucosamine** with ibuprofen or ketoprofen were given.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:880993 CAPLUS
DOCUMENT NUMBER: 134:41096
TITLE: Methods and compounds for the treatment of immunologically-mediated diseases using Mycobacterium vaccae
INVENTOR(S): Watson, James D.; Tan, Paul L. J.; Prestidge, Ross L.
PATENT ASSIGNEE(S): Genesis Research & Development Corporation Limited, N. Z.
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000074715	A1	20001214	WO 2000-NZ85	20000601 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6350457	B1	20020226	US 1999-449013	19991124 <--
CA 2374256	A1	20001214	CA 2000-2374256	20000601 <--
EP 1181051	A1	20020227	EP 2000-937399	20000601 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, MC, PT, IE, SI, LT, LV, FI, RO				
BR 2000011239	A	20020402	BR 2000-11239	20000601 <--
JP 2003501400	T	20030114	JP 2001-501249	20000601

AU 779163	B2	20050106	AU 2000-52579		20000601
US 6723327	B1	20040420	US 2000-710425		20001108
PRIORITY APPLN. INFO.:			US 1999-137112P	P	19990602
			US 1999-449013	A	19991124
			WO 2000-NZ85	W	20000601

AB Methods for the prevention and treatment of disorders, including disorders of the respiratory system, such as infection with mycobacteria such as (M. tuberculosis) or (M. avium), sarcoidosis, **asthma**, allergic rhinitis and lung cancers are provided, such methods comprising administering a composition comprising at least one derivative of delipidated and deglycolipidated (M. vaccae) cells.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:723148 CAPLUS
DOCUMENT NUMBER: 131:346485
TITLE: Methods of screening for agonists and antagonists of the interaction between the human KIAA0001 receptor and ligands thereof
INVENTOR(S): Ames, Robert S.; Romanic, Arnold Anne; Chambers, Jonathan K.; Foley, James J.; Sarau, Henry M.; Stewart, Brian R.
PATENT ASSIGNEE(S): SmithKline Beecham Corporation, USA; SmithKline Beecham PLC
SOURCE: PCT Int. Appl., 42 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
WO 9957245	A1	19991111	WO 1999-US9549		19990430	<--
W: JP						
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE						
EP 1073717	A1	20010207	EP 1999-921581		19990430	<--
R: BE, CH, DE, DK, FR, GB, IT, LI, NL						
US 6238873	B1	20010529	US 1999-303524		19990430	<--
JP 2003524372	T	20030819	JP 2000-547201		19990430	
PRIORITY APPLN. INFO.:			US 1998-83957P	P	19980501	
			WO 1999-US9549	W	19990430	

AB Disclosed are methods for discovering agonists and antagonists of the interaction between UDP-glucose, UDP-galactose, UDP-glucuronic acid, UDP-N-acetyl **glucosamine**, as well as related UDP sugars, and their cellular receptor, human KIAA0001, which may have utility in the treatment of several human diseases and disorders, including, but not limited to: infections such as bacterial, fungal, protozoan and viral infections, particularly infections caused by HIV-1 or HIV-2; pain; cancers; diabetes, obesity; anorexia; bulimia; **asthma**; Parkinson's disease; acute heart failure; hypotension; hypertension; urinary retention; osteoporosis; angina pectoris; myocardial infarction; restenosis; atherosclerosis; diseases characterized by excessive smooth muscle cell proliferation; aneurysms; wound healing; diseases characterized by loss of smooth muscle cells or reduced smooth muscle cell proliferation; stroke; ischemia; ulcers; **asthma**; allergies; benign prostatic hypertrophy; migraine; vomiting; psychotic and neurol. disorders, including anxiety, schizophrenia, manic depression, depression, delirium, dementia, and severe mental retardation; degenerative diseases,

such as neurodegenerative diseases and dyskinesias, such as Huntington's disease or Gilles de la Tourette's syndrome, among others.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:672461 CAPLUS
DOCUMENT NUMBER: 132:2741
TITLE: Resting and cytokine-stimulated human small airway epithelial cells recognize and engulf apoptotic eosinophils
AUTHOR(S): Walsh, Garry M.; Sexton, Darren W.; Blaylock, Morgan G.; Convery, Catherine M.
CORPORATE SOURCE: Department of Medicine & Therapeutics, University of Aberdeen Medical School, Aberdeen, AB25 2ZD, UK
SOURCE: Blood (**1999**), 94(8), 2827-2835
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Eosinophils, which are prominent cells in asthmatic inflammation, undergo apoptosis and are recognized and engulfed by phagocytic macrophages in vitro. We have examined the ability of human small airway epithelial cells (SAEC) to recognize and ingest apoptotic human eosinophils. Cultured SAEC ingested apoptotic eosinophils but not freshly isolated eosinophils or opsonized erythrocytes. The ability of SAEC to ingest apoptotic eosinophils was enhanced by interleukin-1α (IL-1α) or tumor necrosis factor α (TNFα) in a time- and concentration-dependent fashion. IL-1α was found to be more potent than TNFα and each was optimal at 10-10 mol/L, with a significant effect observed at 1 h postcytokine incubation that was maximal at 5 h. IL-1α stimulation not only increased the number of SAEC engulfing apoptotic eosinophils, but also enhanced their capacity for ingestion. The amino sugars **glucosamine**, n-acetyl **glucosamine**, and galactosamine significantly inhibited uptake of apoptotic eosinophils by both resting and IL-1α-stimulated SAEC, in contrast to the parent sugars glucose, galactose, mannose, and fucose. Incubation of apoptotic eosinophils with the tetrapeptide RGDS, but not RGES, significantly inhibited their uptake by both resting and IL-1α-stimulated SAEC, as did monoclonal antibody against αvβ3 and CD36. Thus, SAEC recognize apoptotic eosinophils via lectin- and integrin-dependent mechanisms. These data demonstrate a novel function for human bronchial epithelial cells that might represent an important mechanism in the resolution of eosinophil-induced asthmatic inflammation.
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:126376 CAPLUS
DOCUMENT NUMBER: 128:189187
TITLE: Delivery of nucleic acids to airway epithelial cells as complexes with glycosylated derivatives of polylysine
INVENTOR(S): Glick, Mary Catherine; Scanlin, Thomas F.; Kollen, Wouter J. W.
PATENT ASSIGNEE(S): Children's Hospital of Philadelphia, USA
SOURCE: PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9806869	A1	19980219	WO 1997-US14280	19970813 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5948681	A	19990907	US 1997-907673	19970808 <--
AU 9740659	A	19980306	AU 1997-40659	19970813 <--
PRIORITY APPLN. INFO.:			US 1996-23941P P	19960814
			US 1997-907673 A	19970808
			WO 1997-US14280 W	19970813

AB A method of introducing foreign DNA into animal cells in vivo, especially airway epithelial cells, as a complex with polylysine substituted with glycosyl residues is described. This can be used in methods of treating humans having respiratory disease by gene therapy. The preferred sugar for glycosidation of polylysine is lactose, although α-glucose, β-galactose, mannose mannose-6-phosphate, fucose, or N-acetylglucosamine may also be used. Fusogenic peptides may also be used in the complex to increase the efficiency of uptake. Preparation of a number of glycosylated polylysine derivs. is described. Optimization expts. using cultured CF/T43 cells and a luciferase reporter gene are reported. Binding of the complex to the airway epithelial cells may be by lectins on the surface of the cells.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:745973 CAPLUS
DOCUMENT NUMBER: 128:21880
TITLE: Treatment of **asthma** with TNFR-Ig
INVENTOR(S): Renzetti, Louis Martin
PATENT ASSIGNEE(S): F. Hoffmann-La Roche A.-G., Switz.
SOURCE: PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9741895	A2	19971113	WO 1997-EP2256	19970502 <--
WO 9741895	A3	19980312		
W: AU, BR, CA, CN, CZ, HU, IL, JP, KR, MX, NO, NZ, PL, RU, SG, TR, YU				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2253557	A1	19971113	CA 1997-2253557	19970502 <--
AU 9727764	A	19971126	AU 1997-27764	19970502 <--
AU 725408	B2	20001012		
EP 910413	A2	19990428	EP 1997-921849	19970502 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
CN 1233189	A	19991027	CN 1997-194455	19970502 <--
BR 9708928	A	20000509	BR 1997-8928	19970502 <--
JP 2000510113	T	20000808	JP 1997-539522	19970502 <--
ZA 9703843	A	19971110	ZA 1997-3843	19970505 <--

IN 1997MA00940	A	20050304	IN 1997-MA940		19970505
KR 2000010825	A	20000225	KR 1998-708961		19981106 <--
PRIORITY APPLN. INFO.:			US 1996-16842P	P	19960508
			WO 1997-EP2256	W	19970502

AB The present invention is directed to the preparation of a chimeric TNFα-binding protein which is comprised of the soluble part of the p55 TNF receptor and all domains except the first domain of the heavy chain constant region of a human IgG1 or IgG3 and to the use of such a chimeric TNFα-binding protein for the treatment of **asthma.**

L9 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1995:815245 CAPLUS
DOCUMENT NUMBER: 123:254414
TITLE: Fine specificity of B-cell epitopes on Felis domesticus allergen I (Fel d I): Effect of reduction and alkylation or deglycosylation on Fel d I structure and antibody binding
AUTHOR(S): Vailes, Lisa D.; Li, Ying; Bao, Yongde; DeGroot, Hans; Aalberse, Robert C.; Chapman, Martin D.
CORPORATE SOURCE: Department Medicine, University Virginia, Charlottesville, VA, USA
SOURCE: Journal of Allergy and Clinical Immunology (1994), 93(1, Pt. 1), 22-33
CODEN: JACIBY; ISSN: 0091-6749
PUBLISHER: Mosby-Year Book
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The repertoire of B-cell epitopes on the major cat allergen, Fel d I, was analyzed with monoclonal antibodies (MoAbs) in topog. mapping studies and in immunoassays with antigen derived from other cat (Felidae) species. Four essentially non-overlapping epitopes on Fel d I, designated Fd1A to D, were defined by use of 15 anti Fel d I MoAbs in cross-inhibition RIA. Only MoAbs directed against epitope Fd1B bound to putative Fel d I homologs in hair and dander exts. from 7 other feline species (Panthera species, Leptailurus serval, and Leopardus pardalus). Quant. monosaccharide anal. showed that Fel d I was a glycoprotein, containing high levels of fucose, as well as **glucosamine**, galactose, and mannose. Binding of MoAbs and human IgG or IgE antibody to native, reduced and alkylated or deglycosylated Fel d I was compared by immunopptn. and immunoassay, and the effects of these treatments on the structure of Fel d I were analyzed by SDS-PAGE. On reduction and alkylation, Fel d I dissociated into 14 kDa and 3.2 kDa peptides, and deglycosylation with trifluoromethane sulfonic acid produced a 12-14 kDa peptide. These procedures resulted in a 100-1000-fold loss in murine or human antibody binding activity and caused loss of secondary structure, as judged by CD spectroscopy. Treatment with potassium hydroxide also caused a marked loss in antigenic reactivity. In contrast, enzymic deglycosylation generated a 9 kDa peptide, which showed strong reactivity with murine and human antibodies, comparable to native Fel d I. Thus, MoAbs define a broad repertoire of B-cell epitopes on Fel d I, one of which is expressed by other cat species. These epitopes are conformational and do not appear to involve oligosaccharide residues.

L9 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1992:631970 CAPLUS
DOCUMENT NUMBER: 117:231970
TITLE: Interaction of lectins with human IgE: IgE-binding property and histamine-releasing activity of twelve plant lectins
AUTHOR(S): Shibasaki, Masano; Sumazaki, Ryo; Isoyama, Shigemi; Takita, Hitoshi
CORPORATE SOURCE: Inst. Clin. Med., Univ. Tsukuba, Tsukuba, Japan

SOURCE: International Archives of Allergy and Immunology (1992), 98(1), 18-25
CODEN: IAAIEG; ISSN: 1018-2438
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The IgE-binding reaction and the histamine-releasing response of basophils were examined to a panel of 12 lectins: Con A, Lens culinaris hemagglutinin (LcH), Pisum sativum agglutinin (PSA), wheat germ agglutinin (WGA), soybean agglutinin (SBA), Bauhinia purpurea agglutinin (BPA), peanut agglutinin (PNA), Ricinus communis agglutinin I (RCA-I), Lotus tetragonolobus agglutinin (Lotus A), Ulex europeus agglutinin I (UEA-I), phytohemagglutinin E (PHA-E) and phytohemagglutinin L (PHA-L). IgE from allergic patients bound with high affinity to Con A, LcH, PSA, RCA-I and PHA-E, and with lower affinity to WGA, BPA, Lotus A and UEA-I, but they did not bind to SBA, PNA or PHA-L. There was no apparent individual difference in the reactivity of IgE to these lectins among 10 IgE prepns. from allergic patients. The binding to these lectins, except Lotus A and UEA-I, were competitively inhibited by the lectin-specific sugars or glycopeptide. Upon stimulation by Con A, LcH, PSA, WGA, RCA-1 and PHA-E, leukocytes from allergic patients showed a release of histamine, but cells from IgE-deficient subjects did not respond to these lectins. The histamine-releasing responses by these lectins were also inhibited by specific sugars or glycopeptides.

L9 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1992:15822 CAPLUS
DOCUMENT NUMBER: 116:15822
TITLE: Multifunctional pharmaceutical compounds and methods of use
INVENTOR(S): Glasky, Alvin J.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9114434	A1	19911003	WO 1991-US2066	19910326 <--
W: AU, BR, CA, FI, HU, JP, KR, MC, NO, SU				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
US 5091432	A	19920225	US 1990-500789	19900328 <--
IL 97638	A	19990312	IL 1991-97638	19910321 <--
CA 2079342	A1	19910929	CA 1991-2079342	19910326 <--
CA 2079342	C	20011023		
AU 9176781	A	19911021	AU 1991-76781	19910326 <--
EP 522082	A1	19930113	EP 1991-908058	19910326 <--
EP 522082	B1	19970702		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 05508390	T	19931125	JP 1991-507602	19910326 <--
JP 3198335	B2	20010813		
AT 154880	T	19970715	AT 1991-908058	19910326 <--
ES 2104700	T3	19971016	ES 1991-908058	19910326 <--

PRIORITY APPLN. INFO.: US 1990-500789 A 19900328
WO 1991-US2066 A 19910326

AB Multifunctional pharmaceuticals comprise ≥2 biol. active chemical groups linked by a chemical bridging group. The compds. are useful for treating degenerative diseases and interrelated physiol. systems. 3-(1,6-Dihydro-6-oxo-9H-purin-9-yl)propanoic acid (AlT-0080) enhanced T-lymphocyte proliferation at a moderate dosage (10 μg/mL), yet

enhanced B-lymphocyte function at relatively low dosage (1 μg/mL). Addnl., AIT-0080 enhanced memory function as well as locomotor activity at 0.5 mg/kg in vivo. AIT-0080 was prepared from adenine in 3 steps.

L9 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1987:552353 CAPLUS
DOCUMENT NUMBER: 107:152353
TITLE: An epitope residing in carbohydrate chains of a sea squirt antigen termed Gi-rep
AUTHOR(S): Oka, S.; Shigeta, S.; Ono, K.; Jyo, T.
CORPORATE SOURCE: Fac. Eng., Hiroshima Univ., Higashi-Hiroshima, Japan
SOURCE: Journal of Allergy and Clinical Immunology (1987), 80(1), 57-63
CODEN: JACIBY; ISSN: 0091-6749
DOCUMENT TYPE: Journal
LANGUAGE: English
AB O-glycosidically linked oligosaccharides (Gp-1β-b) were liberated from a large size glycopeptide (GP) fraction (Gp-1) in a Pronase digest of a sea squirt antigen termed Gi-rep by the treatment with 0.1 N NaOH per 1 mol/L of $NaBH_4$. Gp-1β-b as well as Gp-1 and the intact antigen was capable of inducing erythema in the skin of patients with **asthma** with sea squirt allergy. N-glycoside GP fractions (Gp-1β-a and Gp-2β) having the allergenic activity were also prepared as alkali-resistant GPs from Gp-1 and the other relatively small size GP fraction (Gp-2) of the Pronase digest, resp., after the alkali treatment. Chemical, the 3 allergenically active prepns. were rich in galactosamine, **glucosamine**, and fucose in common, although the N-linked carbohydrates were much larger in size than O-linked carbohydrates. Accordingly, it has been expected that the epitope of the sea squirt antigen corresponds to certain oligosaccharide units residing in both of the O- and N-linked carbohydrate chains of the antigen. This suggestion was consistent with the observation that the allergenic activity of Gi-rep was unstable to the periodate oxidation but substantially stable not only to acid, alkali, and heat treatments but also to the enzymic proteolysis with Pronase E.

L9 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1979:401573 CAPLUS
DOCUMENT NUMBER: 91:1573
TITLE: Purification, properties, and analysis of human asthmatic bronchial mucin
AUTHOR(S): Feldhoff, Pamela A.; Bhavanandan, Veerasingham P.; Davidson, Eugene A.
CORPORATE SOURCE: Milton S. Hershey Med. Cent., Pennsylvania State Univ., Hershey, PA, 17033, USA
SOURCE: Biochemistry (1979), 18(11), 2430-6
CODEN: BICHAW; ISSN: 0006-2960
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A high-mol.-weight, mucin-type glycoprotein was isolated from a sample of human bronchial secretion obtained from an asthmatic patient. The glycoprotein elutes in the void volume of a Sepharose 4B column, and its mobility is unchanged in the presence of dithiothreitol. Examination of the material in the anal. ultracentrifuge under equilibrium conditions gave an estimated min. mol. weight of 1.8 + 106 with aggregation to ≥10 + 106. Anal: showed the predominant amino acids to be serine, threonine, and proline with a low content of methionine and cysteine; **glucosamine** and galactosamine were present in approx. equimolar amts. and comprised 28% by weight of the glycoprotein. Composition anal. after alkaline borohydride treatment showed that the saccharide chains were O-glycosidically linked through N-acetylgalactosamine to both serine and threonine residues in the peptide backbone. Carbohydrate anal. by gas

chromatog. identified galactose, fucose, **glucosamine**, galactosamine, and sialic acid in an approx. molar ratio of 3:3:2:2:1. The sialic acid is present as N-acetylneuraminic acid. A portion (7%) of the saccharides are present as galactosyl→N-acetylgalactosaminyl residues linked to the protein core. A glycopeptide fraction was isolated following Pronase digestion and had a mol. weight of 1.5 + 105. This value was not significantly changed by either removal of sialic acid or exposure to guanidinium chloride. These data support the presence of large clusters of oligosaccharides which are covalently linked to the serine and threonine residues of the peptide.

L9 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1954:65244 CAPLUS
DOCUMENT NUMBER: 48:65244
ORIGINAL REFERENCE NO.: 48:11614b-c
TITLE: Plasma **glucosamine** and its pathological variations
AUTHOR(S): Diaz, C. Jimenez; Aguirre, M.; Arjona, E.
CORPORATE SOURCE: Univ. Madrid
SOURCE: Rev. clin. espan. (**1954**), 52, 374-81
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The total **glucosamine** content of human plasma, as determined by the method of West and Clarke (C.A. 32, 7107.2), is normally 75-100 mg. %. In various diseases in which there is increased destruction of tissue (infections, neoplasms) and in collagen diseases, particularly in rheumatic disease, rheumatoid arthritis, and chronic infectious **asthma**, it is increased. Stress also causes an increase in blood **glucosamine.** Substances which offer protection against stress (ACTH, cortisone, N mustards) cause a decrease. The determination of blood **glucosamine** should be an aid in guiding the treatment of cardio-articular rheumatic conditions.

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